EDITION 3

SHOULDER PAIN

RENE CAILLIET, M.D. / PAIN SERIES

SHOULDER
PAIN

Digitized by the Internet Archive
in 2021 with funding from
Kahle/Austin Foundation

https://archive.org/details/shoulderpain0000cail

SHOULDER PAIN

EDITION 3

RENE CAILLIET, M.D.

Professor and Chairman Emeritus
Department of Physical Medicine and Rehabilitation
School of Medicine
University of Southern California
Los Angeles, California

Director
Rehabilitation Services
Santa Monica Hospital Medical Center
Santa Monica, California

Illustrations by R. Cailliet, M.D.

F.A. DAVIS COMPANY • Philadelphia

Other titles by Rene Cailliet from F.A. Davis Company:

Foot and Ankle Pain
Hand Pain and Impairment
Knee Pain and Disability
Low Back Pain
Neck and Arm Pain
Scoliosis
The Shoulder in Hemiplegia
Soft Tissue Pain and Disability

Copyright © 1991 by F.A. Davis Company

Copyright 1981 and 1966 by F.A. Davis Company. All rights reserved. This book is protected by copyright. No part of it may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without written permission from the publisher.

Printed in the United States of America
Last digit indicates print number 10 9 8 7 6 5 4 3

NOTE: As new scientific information becomes available through basic and clinical research, recommended treatments and drug therapies undergo changes. The author and publisher have done everything possible to make this book accurate, up-to-date, and in accord with accepted standards at the time of publication. The author, editors, and publisher are not responsible for errors or omissions or for consequences from application of the book, and make no warranty, expressed or implied, in regard to the contents of the book. Any practice described in this book should be applied by the reader in accordance with professional standards of care used in regard to the unique circumstances that may apply in each situation. The reader is advised always to check product information (package inserts) for changes and new information regarding dose and contraindications before administering any drug. Caution is especially urged when using new or infrequently ordered drugs.

Authorization to photocopy items for internal or personal use, or the internal or personal use of specific clients, is granted by F.A. Davis Company for users registered with the Copyright Clearance Center (CCC) Transactional Reporting Service, provided that the fee of $.10 per copy is paid directly to CCC, 27 Congress St., Salem, MA 01970. For those organizations that have been granted a photocopy license by CCC, a separate system of payment has been arranged. The fee code for users of the Transactional Reporting Service is 8036-1614/910 + $.10.

Preface to Third Edition

Shoulder pain is increasingly presenting to the clinician as a significant disabling musculoskeletal disability. There are increasing numbers of patients suffering from injuries incurred while exercising to maintain health—to decrease cardiovascular disease, control arteriosclerotic vascular disease as an adjunct to diet, remain fit by aerobic contortions, and to maintain ideal body weight.

Exercise that has been advocated to accomplish all the above health benefits have included upper extremity exercise and forceful athletic activities. The new orthopedic specialty, sports medicine, has elevated the pathologic shoulder to the expertise of the family practitioner for diagnosis and to the orthopedic sports medicine surgeon for cure.

Many of the rheumatologic diseases have included the shoulder to a greater degree; as it has interested the neurologist in the care of the stroke patient, the physical therapist and the occupational therapist have now concentrated on the shoulder with greater interest.

The psychological implications in impaired and painful neuromuscular shoulder dysfunction are explored for their full importance. The modalities used in this extremity, as in the treatment of the low back and the neck, have progressed to greater understanding of soft tissue pathology, functional anatomy, kinesthetics, and especially neuromuscular physiology. The neurologic control of proper musculoskeletal function and malfunction is emphasized.

In this updated edition, newer concepts are reviewed as newer treatments are discussed and evaluated. The hemiplegic shoulder is included in its entirety. Reflex sympathetic dystrophy is discussed in a full chapter, as it has become a serious yet often overlooked disease entity.

The shoulder in sports is given greater emphasis than before since it has become a prominent source of painful disabling soft tissue pathology.

Since a major function of the shoulder is to place the hand in functional positions, the shoulder must be examined in impaired painful hand functions. Its direct musculoskeletal and neurologic relationship to the cervical spine mandates its being fully evaluated in ascertaining accurate diagnoses and appropriate subsequent treatment.

The enlarged, updated reference lists throughout the text indicate the increasing importance of this aspect of neuromusculoskeletal functional anatomic entity.

RENE CAILLIET, M.D.

Preface to Second Edition

Pain in the shoulder region is exceeded in clinical frequency only by pain in the low back and pain in the neck. Recent developments and modifications of older concepts concerning shoulder pain, together with the need for further clarification of associated disease syndromes, have prompted this new edition.

The shoulder remains a complex functional unit with numerous tissues capable of causing joint dysfunction. As in all musculoskeletal systems, a thorough knowledge of functional anatomy is mandatory, and the examiner must evaluate every aspect of this functional anatomy. Treatment must, therefore, be based on modification or correction of these malfunctions. The shoulder joints, some eight or nine in number, may all contribute to pain and dysfunction, thus methodical examination, individually and collectively, of all these joints must be performed.

When man began to assume the upright position, and his forelegs became arms and hands, the shoulder and its arm components became useful to place the hand in functional position and to acquire greater mobility at the expense of stability. Due to this lack of stability, degeneration, damage, pain, and malfunction can result.

This new edition is meant to supplement the previous one. Its organization is basically the same in that functional anatomy is stressed, and examination to determine any deviation from normal can be appreciated. The patient's history and examination thus become meaningful, and treatment evolves from this concept, based on physiologic principles.

New chapters have been added that involve the hemiplegic shoulder and the shoulder-hand-finger syndrome, both of which are seen constantly in daily practice. The bibliography has been chosen to update current references to the concepts postulated.

RENE CAILLIET, M.D.

Contents

List of Illustrations .... **xiii**

Introduction .... **xix**

CHAPTER 1. Functional Anatomy **1**
- Mechanics of Joints 4
- The Glenohumeral Joint 6
- Glenoid Fossa 11
- Glenohumeral Capsule 12
- The Suprahumeral Joint 16
- Musculature of the Glenohumeral Joint 18
- Muscle Spindle System 18
- The Rotator Cuff 22
- The Rotator Cuff Tendon 25
- The Deltoid Muscle 26
- Glenohumeral Movement 27
- Scapular Component of the Scapulohumeral Rhythm 33
- Acromioclavicular and Sternoclavicular Joint Motion 38
- Scapulohumeral Rhythm Movement 42
- The Biceps Mechanism 46
  - Bibliography 50

CHAPTER 2. Tissues Sites and Mechanisms of Shoulder Girdle Pain **51**
- Tendinitis: Trauma, Attrition, and Degeneration 54
- Symptoms of Supraspinatus Tendinitis 60
- Calcific Tendinitis 63
- Bursitis: Subdeltoid (Subacromial) Bursitis 67

Diagnosis: Supraspinatus Tendinitis (Subdeltoid Bursitis) 68
Treatment 73
Therapeutic Exercises 81
Strengthening Exercises 84
Physiologic Effect of Exercise 87
Bibliography 93

CHAPTER 3. Rotator Cuff Tear: Partial and Complete 95
Physical Findings 97
Partial and Complete Tear 97
Confirmatory Test of Cuff Tear 99
Treatment of the Rotator Cuff Tear 100
Bibliography 103

CHAPTER 4. The Frozen Shoulder: Adhesive Capsulitis, Adhesive Bursitis 105
Clinical Picture: Physical Findings 6
Radiologic Signs 8
Treatment of the Frozen Shoulder 8
Conditions Relating to the Frozen Shoulder 9
Painful Shoulder Problems 10
Suggested Treatments 13
Technique of Manipulation 17
Distention Arthrography: Brisement 18
Bibliography 19

CHAPTER 5. Posture in Shoulder Pain 124
Posture 126
Posture Development 127
Bibliography 134

CHAPTER 6. Trauma to the Shoulder 135
Dislocation 135
Mechanism of Dislocation 137
Diagnosis of Dislocation 141
Treatment of Dislocation 142
Acromioclavicular Joint Injury 144
Diagnosis 144
Clavicular Fractures 147
Bibliography 147

CHAPTER 7. Sports Injuries to the Shoulder 149
Treatment of the Injured Athlete 157
External Rotator Exercise 167

CHAPTER 8. Neurologically Referred Shoulder Pain 168
- Cervical Nerve Root Involvement (Radiculopathy) ... 168
- Mechanism of Cervical Radicular Pain in the Upper Extremity ... 172
- Thoracic Outlet Syndrome ... 176
  - Symptoms of TOS ... 180
  - Cervical Rib ... 182
  - Scalene Anticus Syndrome ... 182
  - Claviculocostal Syndrome ... 183
  - Hyperabduction TOS Syndrome ... 183
  - Physical Examination ... 183
  - Treatment ... 185
- Brachial Plexus Injuries ... 186
  - Brachial Plexus Neuritis ... 188
- Suprascapular Nerve Entrapment ... 189
- Dorsal Scapular Nerve Entrapment ... 191
    - Bibliography ... 191

CHAPTER 9. The Shoulder in the Hemiplegic Patient 193
- The Completed Stroke ... 193
  - Transient Ischemic Attack (TIA) ... 194
  - Flaccid Stage ... 194
  - Spastic Stage ... 195
  - Synergy Stage ... 198
- The Shoulder in All Stages of Hemiplegia ... 198
- Pathomechanics of Shoulder Function in Stroke Stages ... 201
  - Flaccid Stage ... 201
  - Spastic Stage ... 202
  - Synergy Stage ... 205
- Sensory Loss in Stroke ... 205
- Functional Recovery ... 206
- Treatment Concepts of the Hemiplegic Shoulder ... 207
  - Flaccid Stage ... 207
- Subluxation of the Shoulder ... 209
- The Hemiplegic Shoulder in the Spastic Stage ... 214
- Heterotropic Ossification ... 223
  - Treatment ... 224
    - Bibliography ... 225

CHAPTER 10. Reflex Sympathetic Dystrophy 227
- Reflex Sympathetic Dystrophy ... 227
- Reflex Sympathetic Dystrophy in Shoulder Lesions ... 235
- Shoulder-Hand-Finger Syndrome ... 235
  - Arterial Component ... 235
  - Venous Component ... 235

Mechanism of Shoulder-Hand-Finger Syndrome 237
Diagnosis of Shoulder-Hand-Finger-Syndrome 237
Treatment 243
Psychiatric Aspects 249
Bibliography 251

CHAPTER 11. Myofascial Pain: Scapulocostal Syndrome ... 253
Scapulocostal Syndrome 253
Fibrositis and Muscular Referred Pain Syndromes 257
Clinical Management 260
Bibliography 260

CHAPTER 12. Visceral Referred Pain 262
Bibliography 265

Index 267

Illustrations

1– 1 The joints of the shoulder girdle 2
1– 2 The acromiocoracoid arch 4
1– 3 Joint surfaces 5
1– 4 Asymmetrical joint surfaces 5
1– 5 Hydrodynamic lubrication 6
1– 6 Joint motion: spin or rotation 7
1– 7 Joint motion 7
1– 8 Capsular restriction of rotation 8
1– 9 Congruous-incongruous joints 9
1–10 The scapula 10
1–11 The coracohumeral ligament 11
1–12 The acromiocoracoid arch 12
1–13 The glenohumeral synovial capsule 13
1–14 Capsular action during glenohumeral movement 14
1–15 The anterior capsule and the glenohumeral ligaments 15
1–16 Manipulation treatment of involuntary motion of the glenohumeral joint (A through D) 16
1–17 Sites of muscle origin and insertion upon the scapula and the humerus 19
1–18 The supraspinatus muscle and the infraspinatus muscle 20
1–19 Spindle system function 21
1–20 Intrafusal muscle spindle 22
1–21 Capsular-passive cuff support 23
1–22 Rotator cuff insertion upon the humerus 24
1–23 The subscapularis muscle 25
1–24 Circulation of the tendons of the cuff: the *critical zone* 26
1–25 Blood circulation 27
1–26 The deltoid muscle and its isolated function 28
1–27 The planes of arm movement 29

1–28 Function of the supraspinatus muscle 30
1–29 Combined cuff and deltoid action upon the glenohumeral articulation 31
1–30 Abduction angle of deltoid 32
1–31 Rotator cuff mechanism 32
1–32 Supraspinatus external rotation of the humerus 33
1–33 Scapular musculature: rotators 34
1–34 Downward rotators of the scapula 35
1–35 Latissimus dorsi and pectoralis muscle attachment upon the humerus 37
1–36 Pectoralis major 38
1–37 Evolution of the acromioclavicular disk (meniscus) 39
1–38 The acromiocoracoid arch 39
1–39 Action of the coracoclavicular ligaments upon the acromioclavicular joint 40
1–40 Static support of the scapula by claviculoscapular ligaments 41
1–41 Scapular elevation resulting from clavicular rotation 42
1–42 The sternoclavicular joint 43
1–43 Muscles acting upon the clavicle 44
1–44 Scapulohumeral rhythm 45
1–45 Deltoid action upon the glenohumeral joint 46
1–46 Accessory movement of the scapulohumeral rhythm other than the glenohumeral movement 47
1–47 Biceps mechanism 49

2– 1 Sites of tissue pain 53
2– 2 Tissues within the suprahumeral joint space 55
2– 3 Roentgenographic changes in shoulder dysfunction 57
2– 4 Forward-head posture 58
2– 5 Faulty occupational posture: strain from sitting 59
2– 6 Faulty occupational posture: strain from standing 60
2– 7 Chemical mediation of chemical nociceptors from trauma 61
2– 8 Vascular sequelae of tissue trauma 61
2– 9 Sequence of tendon degeneration 62
2–10 Postural effect upon glenohumeral range of motion 64
2–11 Natural sequence of calcific tendinitis 65
2–12 Evolution of the calcified tendinitis 67
2–13 Subacromial bursa 68
2–14 Acute tendinitis and inflammation of contiguous tissues 69
2–15 Trigger points 70
2–16 Mechanism by which irritation leads to functional disability 72
2–17 Shrugging mechanism 73
2–18 Painful arc 74

2–19 Collagen fiber intersectional gliding 75
2–20 Codman pendular exercise 77
2–21 Active pendular glenohumeral exercise 78
2–22 Sites of suprahumeral injections 80
2–23 Suprascapular nerve block 82
2–24 Exercises (A through H) 83
2–25 Home exercise to increase shoulder range of motion 85
2–26 External range of motion exercise 86
2–27 Exercise to stretch anterior capsule and to increase posterior flexion 87
2–28 Overhead exercise 88
2–29 Overhead range of motion exercise 89
2–30 External rotator strengthening (supraspinatus muscle) 90
2–31 Correct and incorrect use of "wall climbing" exercise 91
2–32 Scapular mobility exercises 92

3– 1 Cuff-tear 96
3– 2 Cuff-tear 98
3– 3 Drop arm test for rotator cuff tear 100
3– 4 Forearm drop test for rotator cuff tear 101
3– 5 Injection technique for intra-articular arthrogram and brisement treatment 101
3– 6 Sites of acromionectomy 102
3– 7 Proper spica cast for the torn cuff treatment 103

4– 1 Tissue sites within the glenohumeral joint space for formation of "adhesions" 106
4– 2 Schematic stages toward functional disability in the painful shoulder 107
4– 3 Periarticular case of the suprahumeral joint 109
4– 4 Subtle signs of shoulder limitation 111
4– 5 Adhesive capsulitis 113
4– 6 Manipulation treatment of *involuntary* motion of the glenohumeral joint (A through D) 115
4– 7 Rhythmic stabilization manipulation of the glenohumeral joint 117
4– 8 Schematic graph of rhythmic stabilization 118
4– 9 Antigravity treatment of the edematous extremity 119
4–10 Home exercises for painful restricted shoulder 120
4–11 Shoulder exercises 121

5– 1 The shoulder as related to posture 125

5– 2 Comic spoof of erect posture 126
5– 3 Chronic development of cervical lordosis 127
5– 4 Neurologic concept of posture 129
5– 5 Gravity effect on forward-head posture 132
5– 6 Proper posture exercise 132

6– 1 Anterior capsule, glenohumeral ligaments, and avenue of anterior shoulder dislocation 136
6– 2 Four types of shoulder dislocation 137
6– 3 Relationship of age of patient to mechanism of anterior dislocation 138
6– 4 Mechanism of dislocation 139
6– 5 The planes of arm movement 140
6– 6 Intra-articular fracture in recurrent shoulder dislocation 141
6– 7 Kocher manipulation for closed treatment of dislocation 143
6– 8 Acromioclavicular injuries 144
6– 9 Immobilization of acromioclavicular separation 146

7– 1 Total derotation of trunk and arm during a tennis serve 151
7– 2 Cocking—second stage of throwing mechanism 152
7– 3 Overhead pitching motion: stage 3 and 4 153
7– 4 Apprehension and relocation tests for shoulder subluxation 156
7– 5 Stretch exercise for increased external rotation 157
7– 6 Hyperextension needed for better bowling 158
7– 7 Hyperextension exercise of the shoulder 159
7– 8 Dips exercises for hyperextension of the shoulder posteriorly 160
7– 9 Leg gravity angle during running 161
7–10 Swinging warm-up rotatory trunk flexibility exercise 162
7–11 Trunk twist exercise 163
7–12 Side bending (1) 164
7–13 Side bending (2) 165
7–14 Strengthening exercises for the rotator cuff 166

8– 1 Component fibers of a cervical nerve 169
8– 2 Lateral view of a functional unit of the cervical spine C-3 to C-7 ... 169
8– 3 Direction of foraminal grooves 170
8– 4 Brachial plexus (schematic) 170
8– 5 Sixth cervical nerve root irritation 171
8– 6 Seventh cervical nerve root irritation 172
8– 7 Eighth cervical nerve root irritation 173
8– 8 Regions of shoulder pain referral from cervical radiculitis 174

8– 9 Dermatomes of cervical nerve roots C_5 to C_8 175
8–10 Forminal opening variations 176
8–11 Spurling compression test for radiculitis 177
8–12 Manual traction and arm abduction test for cervical radiculitis 178
8–13 Thoracic outlet 179
8–14 Musculature of the head and neck (A through D) 180
8–15 Anterior view of *prevertebral fascia* and muscles 181
8–16 Scalene anticus syndrome 184
8–17 Claviculocostal and pectoralis minor syndromes 185
8–18 Posture-scapular elevation exercises 187
8–19 Schematic brachial plexus 188
8–20 Suprascapular nerve block 190

9– 1 Central nervous system 194
9– 2 Estimate of spontaneous recovery from stroke 195
9– 3 Midbrain, pons, and medulla 196
9– 4 Spinal and supraspinal motor centers 197
9– 5 Premotor cortex (schematic) 198
9– 6 Flexor synergy of the upper extremity 199
9– 7 Glenoid fossa 199
9– 8 Spindle system control of the supraspinatus 200
9– 9 Scapular depression 202
9–10 Mechanism of glenohumeral subluxation 203
9–11 Internal rotators of the humerus 204
9–12 Spastic medial scapular muscles 205
9–13 Johnstone air sleeve 208
9–14 Exercise 210
9–15 Exercise 211
9–16 Exercise 212
9–17 Proposed design for prevention of subluxation 213
9–18 Rood sling 213
9–19 Shoulder slings 214
9–20 Wheelchair arm sling 215
9–21 Upper illustration depicts the extensor pattern of the upper extremity 216
9–22 Denial demonstrated by patient (drawing) 218
9–23 Patient crawling with weight on affected arm 220
9–24 Patient sitting and leaning on affected arm 221
9–25 Rhythmic stabilization technique to increase shoulder joint range of motion 222

10– 1 Axoplasmic neural transport: A theory 229

10– 2 Axonal outgrowths forming a neuroma (schematic) 231
10– 3 Neuron pathways of pain 232
10– 4 Causalgic (autonomic) transmission of pain sensation 233
10– 5 Postulated neurophysiologic mechanism
of sympathetic maintained pain (SMP) 234
10– 6 Venous lymphatic pumps of the upper extremity 236
10– 7 Sequence leading to frozen shoulder-hand-finger syndrome 238
10– 8 Finger changes in the hand-shoulder syndrome 239
10– 9 Normal flexion-extension of the metacarpophalangeal joints 240
10–10 Hand patterns 242
10–11 Technique of stellate ganglion or brachial plexus block 244
10–12 Antigravity treatment of the edematous extremity 246
10–13 Removal of finger edema 247
10–14 Mobilization of the joints of the wrist and fingers 248
10–15 Manipulation treatment of involuntary motion
of the glenohumeral joint (A through D) 249

11– 1 Levator scapulae trigger zone in postural fatigue syndrome 254
11– 2 Sitting posture to avoid 255
11– 3 Standing posture to avoid 256

12– 1 Visceral sources of shoulder pain 263
12– 2 Neural mechanism of visceral referred pain 264

Introduction

The complaint of pain in the upper extremity can present a confusing problem to the practitioner and to the therapist. The *upper extremity* includes the cervical spine and its joints, ligaments, muscles, and nerves. It contains the shoulder girdle, the upper arm, forearm, wrist, and hand. Pain can originate within any of these structures or be felt there, yet it could originate elsewhere in the body—but felt in the upper extremity.

The differential diagnosis of pain and its etiology remains in an accurate, meaningful case history and a pertinent physical examination. Numerous diagnostic procedures have emerged such as cortical evoked potential, neurologic diagnostic tests, thermography, magnetic resonance images (MRI), computerized scanning procedures (CT), and nuclear bone scannings, to mention a few.

No special test or procedure has replaced a meaningful history or examination. All pertinent information is and must be based on complete knowledge of normal neuromusculoskeletal functional anatomy. Clarification of pathology as *the* cause of the pain and impairment remains the basis for appropriate treatment. Soft tissue remains at the core of impairment even though it is often beyond the scope of many laboratory diagnostic procedures.

Special surgical procedures such as arthroscopic and microscopic procedures have emerged and have been evaluated. Surgery remains important after all other *appropriate* nonsurgical procedures have been exhausted. Abuse or misuse of diagnostic modalities have engendered excessive medical expenses, confusion for the patient population, and have falsely exonerated the prescribing physician and therapist. Chronic pain and unnecessary permanent, partial, or total functional impairment has ensued from failure of an early, correct initial diagnosis followed by proper treatment.

The purpose of this new edition is to provide the clinician with a sound basis for performing a significant meaningful case history and appropriate examination and for prescribing rational treatment for the patient complaining of pain and impairment of the shoulder complex.

CHAPTER 1

Functional Anatomy

The term *shoulder joint* needs clarification if this anatomic site of pain and dysfunction is to be understood properly.

In the common clinical language of shoulder pathology, it is the glenohumeral joint that is emphasized. The glenohumeral joint may be the major site of pain and impaired function in the majority of incidences of shoulder pain, but there are numerous other joints of the shoulder complex that are involved in any upper extremity function. To discuss and understand fully how the upper extremity functions, the term *shoulder girdle complex* is more appropriate.

The other definitions of arm trunk mechanism are thoracoscapulohumeral articulations, shoulder-arm-complex, or merely shoulder girdle. In any function of placing the hand in a meaningful position for manual activities, all joints of the shoulder complex are involved.

In purposeful upper extremity function, every articulation involved must be operant and well coordinated. It becomes apparent that in evaluating normal function, and thus in establishing where abnormality results in pain and dysfunction, every joint must be evaluated. The nerve supply of each muscle must be clarified, and the feedback must be ascertained to assure well-coordinated function. There is no part of the musculoskeletal system in the human body that illustrates more clearly the axiom that proper, efficient function demands complete integrated neuromusculoskeletal-articular function than does the shoulder girdle complex.

When, through evolution, the human being became erect and upright with dexterity of the upper extremity, the upper extremity transformed the appendage from weight bearing to prehension and manipulation. The shoulder girdle thus assumed greater range of motion, replacing stability and force—motion replaced weight bearing and ambulation.

The brain (neocortex) developed accordingly with the premotor cortex enlarging in the regions that control speech, facial movement, and thumb and finger function. The cortical area controlling shoulder function similarly enlarged. Prehension and precise digital manipulation developed in human beings. The shoulder also improved in precise motion, coordination, strength, and endurance, assisting the upper extremity functions.

The arm comprises the upper extremity supported to the trunk by the scapula.

Coordinated motion of the arms with the scapulae as their proximal attachment to the trunk was introduced by E.A. Codman in his classic work, "The Shoulder." He defines the synchronous motion of the arm as *scapulohumeral rhythm.*

The numerous components of the upper extremity are shown in Figure 1–1. They are numbered in this illustration with (1) the glenohumeral joint and

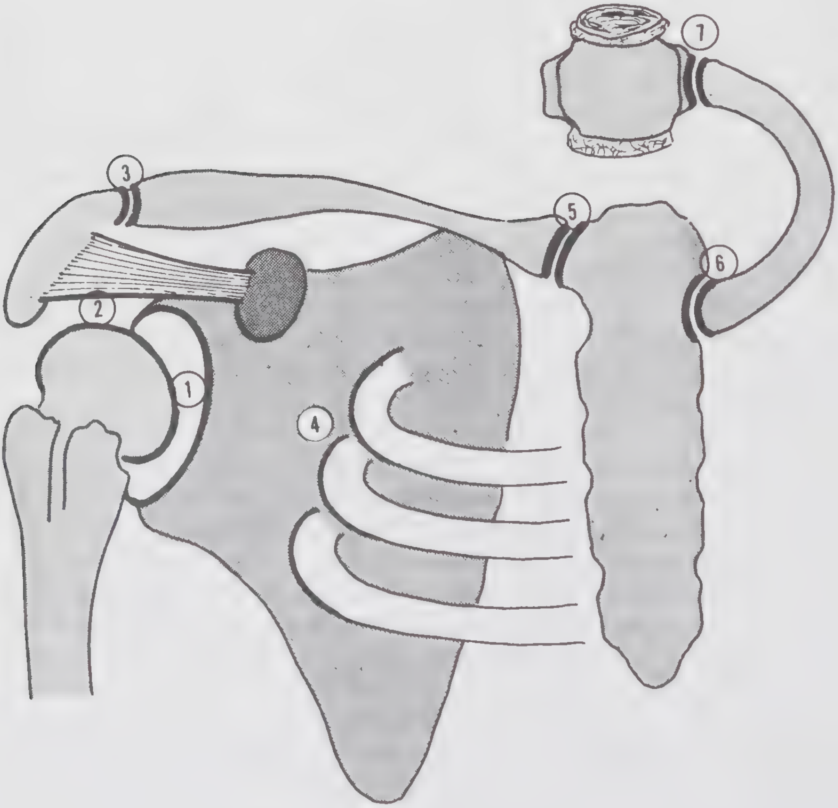


Figure 1–1. The joints of the shoulder girdle: (1) glenohumeral, (2) suprahumeral, (3) acromioclavicular, (4) scapulocostal, (5) sternoclavicular, (6) sternocostal, (7) costovertebral.

the proximal articulation and (7) the costovertebral joint. This number sequence is arbitrary.

Inasmuch as the upper extremity is basically supported by the trunk through these articulations, it is apparent that the upper extremity depends upon soft tissues for support and function: muscles, fascia, ligaments, tendons, and joint capsules.

The proximal joint of the shoulder complex (7) is the *costovertebral joint.* Progressing outward, the next contiguous joint is (6) the *sternocostal joint.* The sternum elevates and lowers through the attachment of the sternum to the vertebral spine by way of the ribs. Hence, rightfully, the sternocostovertebral articulations (6) should also be cited as joints of the shoulder girdle.

The scapula is held away from the vertebral column by a strut, the clavicle. The clavicle is attached to the sternum by (5) the sternoclavicular joint, where motion occurs in numerous directions: elevation, depression, forward and backward motion, and circumduction. Each of the joints within the scapulohumerovertebral complex will be discussed and evaluated separately.

The scapula articulates upon the rib cage at the (4) scapulocostal joint or scapulothoracic joint. This can be termed a *joint* because it meets the definition: where two bones meet to permit motion. The scapulocostal joint meets this definition arbitrarily inasmuch as it is a concaved flat bone that glides upon convex costal bones separated only by muscle, fascia, and bursae. This is, however, a functional articulation, therefore, it is a *joint.*

The major portion of the shoulder girdle complex, the arm portion, begins at (3) the acromioclavicular joint, where the scapula is suspended, rotating to and from the thorax.

The clinically noted *shoulder joint,* the glenohumeral joint, is essentially the proximal joint of the arm and is more complex in structure and function. Separating the head of the humerus from its contiguous scapular component and part of the glenohumeral joint is the *suprahumeral joint.* Although it is not essentially a joint formed by two articulating bones, there is an anatomic relationship that is both functional and vulnerable to a pathologic condition.

Within its glenoid fossal attachment, the head of the humerus is covered by a ligamentous arch, the *coracoacromial ligament* (Fig. 1–2). The ligament between the anteriorly placed coracoid process and the acromial process serves essentially to function as a roof over the glenohumeral joint. There is no movement between these two bones—the coracoid and the acromion—because they are both structures within the scapula. Thus, the function of this ligament is passively covering and protecting the joint from direct trauma.

As the humerus moves during arm motion, it passes directly under the coracohumeral ligament and thus conforms to the definition of a joint.

The glenohumeral joint will be studied in detail here because this joint is the site of major motion and significant pathologic conditions. All its tissues are significant.

The biceps mechanism will be discussed here because the relationship of

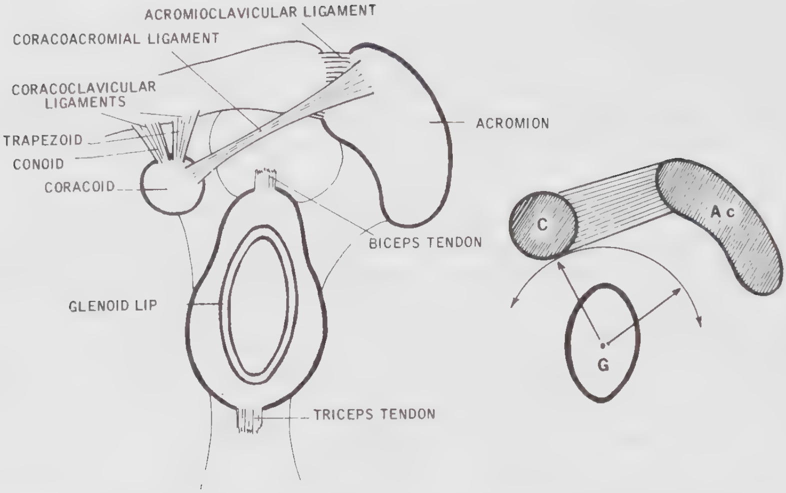


Figure 1–2. The acromiocoracoid arch. The diagram depicts the shape of the glenoid fossa and its relationship to the acromial process, the coracoid process, and the coracoacromial ligament. In essence this diagram shows the socket of the glenohumeral joint and also portrays the relationship of the suprahumeral joint.

the tendon to the glenohumeral joint has functional significance, and it is also a site of pain and pathologic process. It will be discussed along with the other soft tissues involved in the glenohumeral functions.

MECHANICS OF JOINTS

The understanding of joint mechanical function has been furthered by a cooperation between the engineering profession and the field of medicine. Physical treatment of joint dysfunction depends upon a clear understanding of the mechanical function of any joint and its related tissues.

A typical synovial joint is formed by two opposing articular surfaces, each covered by cartilage. It is enclosed within a capsule that contains synovial fluid which is secreted as a lubricant by synovium.

There are essentially two types of joint surfaces: *ovoid* and *sellar* (Fig. 1–3), where the ovoid is uniformly concave or convex, respectively. The curvature of the opposing bone of the articulation is congruent or incongruent, depending upon the arc or curvature and the relationship between the two surfaces (MacConnaill, 1946).

The articulating surfaces of opposing bones comprising a joint are considered perfect curves fitted into each other with equal contact at each point. Motion of this joint type occurs around a fixed axis of rotation.

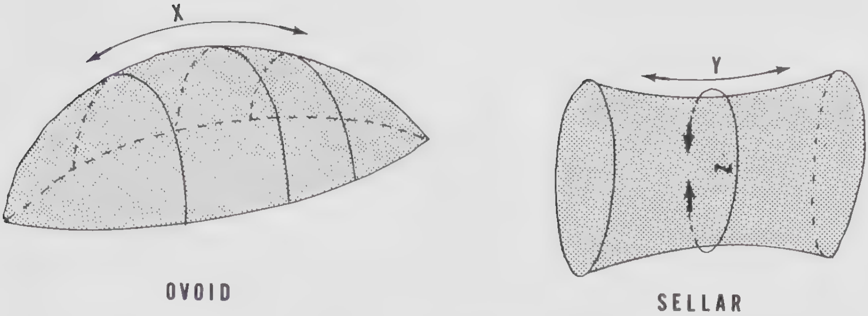


Figure 1–3. Joint surfaces. There are two basic joint surfaces: ovoid and sellar. The ovoid is uniformly convex (X) at each point along the surface. The sellar surface is convex (Z) in one plane and concave (Y) in the perpendicular plane.

This definition of a *true congruous joint* is contrary to accepted engineering principles. Engineering studies of joints have shown that articular surfaces are variable rather than uniform. A true congruous joint would not permit lubrication of synovium, whereas a degree of incongruity will move the lubricant to either side of the joint (Fig. 1–4).

A true congruous joint implies direct contact of the articular surfaces at every point around the curvatures of the end surfaces. This contact creates a *close packed* (MacConnaill, 1951) relationship and would *bind* the joint. No lubrication would evolve (Fig. 1–5).

In an incongruous joint, the articulating surfaces touch at varying sites and cover a small area. The remainder of the joint space is separated to a greater degree (see Fig. 1–5).

In the human body only the hip joint (femoral head into the acetabulum)

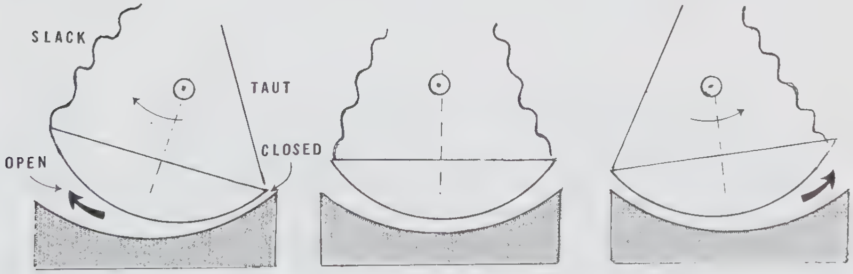


Figure 1–4. Asymmetrical joint surfaces. The asymmetrical joint surfaces of incongruous joints cause synovial fluid *(large arrow)* to flow toward the open articular area. The joint ligaments remain taut on the closing side and become slack on the opening side.

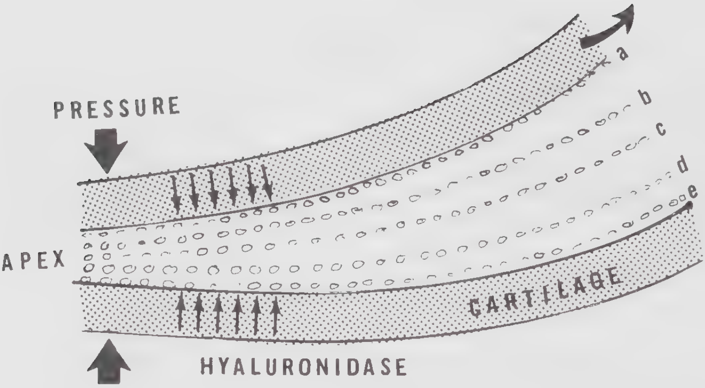


Figure 1–5. Hydrodynamic lubrication. Nonparallel joint surfaces form a wedge-shaped lubricating fluid, some of which stays at the apex. The lubricating fluid moves in layers—a, b, c, d, e—at the same speed as the articulating bone, but a layer (a–e) adheres to both articular surfaces. A shearing force between layers causes deformation of the fluid.

The lubricant is both adhesive and viscous, being coated by hyaluronic acid, which is created by the synovium and cartilage. Even without movement a layer(s) remains between the two opposing joint surfaces.

approximates a congruous joint. This is the joint of greatest stability and yet it is limited in motion. In this (congruous) joint the head of the femur is deeply seated within the acetabulum. The axis of rotation remains essentially in the center, and rotation (flexion, extension, abduction, adduction, and internal-external rotation) revolves around this fixed axis centrum.

Motion of an incongruous joint is that of *spin* rather than roll or rotate (Fig. 1–6). If there is spin of the articulating joint surfaces combined with rotation, which is what most joints do, the end result is known as arc-slide (Fig. 1–7).

The capsule of a congruous joint is limited, and it shortens with rotation. A portion of the capsule may be thickened in the portion that becomes extended and may act to restrict motion in that direction. This thickened portion of the capsule literally becomes a ligament (Fig. 1–8).

THE GLENOHUMERAL JOINT

The glenohumeral joint shown as (1) in Figure 1–1 has been alluded to as the principal joint involved in shoulder pain and dysfunction.

The glenohumeral joint—the articulation between the rounded convex head of the humerus and the concave surface of the shallow glenoid fossa—is a classic example of an incongruous joint, both structurally and functionally.

To further discuss an incongruous joint and to apply this definition to the glenohumeral joint, several aspects of incongruency must be repeated. The ar-

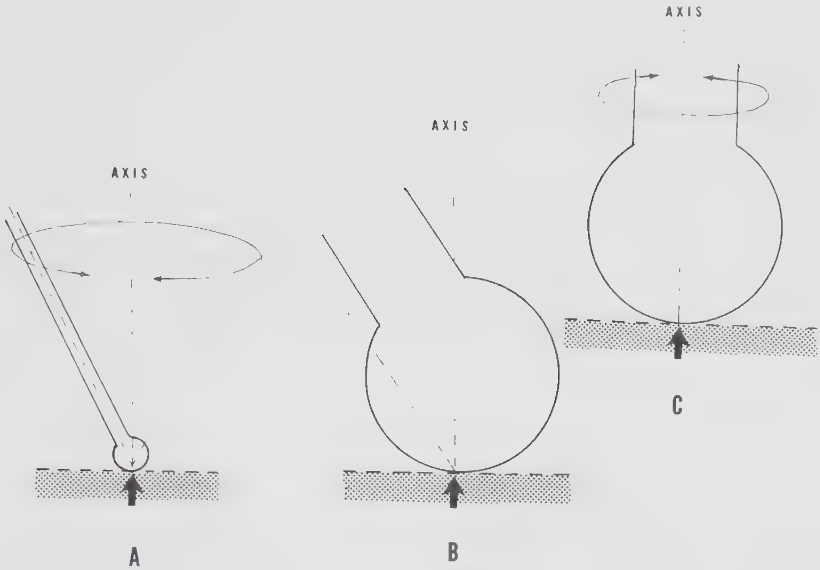

AXIS
AXIS
AXIS
A
B
C

Figure 1–6. Joint motion: spin or rotation. True spin (C) is exemplified in a spinning top about one point. If there is a change of the axis perpendicular (A) to the surface during spinning, a *spin rotation* occurs (B).

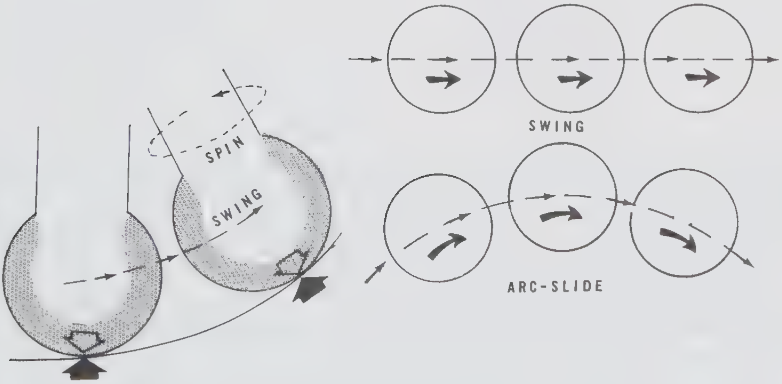

SPIN
SWING
SWING
ARC-SLIDE

Figure 1–7. Joint motion. Slide of a joint moving in one plane is termed *swing*. In this motion there is no rotation or spin. If there is simultaneous spin, the motion is termed *arc-slide.*

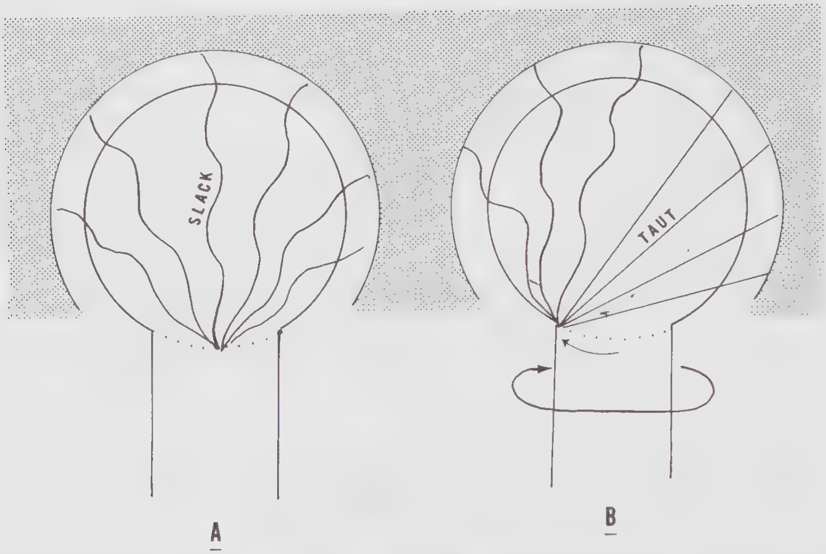


Figure 1–8. Capsular restriction of rotation. Figure *(A)* is the neutral position of a joint with the capsule generally slack. When there is rotation of the bone *(B)*, the capsule becomes taut and limits motion. The limiting capsular fibers may be enlarged enough to function as a *ligament.*

ticulating surfaces are not symmetrical surfaces; the humeral head is more convex, and the glenoid fossa is a shallower concavity than is relevant in the femoral head–acetabular relationship.

As the head of the humerus (convex) moves, it does so in a downward, anterior, posterior, inversion, eversion direction in a gliding manner, as dictated by the expected motion of the arm.

The center of rotation in motion of the humeral-glenoid joint (incongruous) changes, whereas in the femoral acetabular motion (congruous) there is minimal, if any, change. As the center of rotation changes, the action of the muscles varies about this axis. In the congruous joint the flexor and the extensor, internal and external rotators *all* move the part around the fixed center. Muscular action is relatively simple, inasmuch as it is for motion rather than for support. The support of the congruous joint is the deep-seated relationship between the male and female aspects of the articular surfaces.

In the incongruous joint there is no deep seating, and thus the muscles must furnish support while the head moves simultaneously around the changing center of rotation. This mandates a dual action of muscles—support and motion.

As the capsule is attached about the circumference of the concave socket and about the convex contour of the head and shaft of the humerus (femur) in the congruous joint, the capsule is the same length throughout, and it shortens symmetrically with motion. In the incongruous joint the capsule is longer in one aspect and shorter in the other, because the points of attachment differ as the head rotates (Fig. 1–9).

The socket of the incongruous glenohumeral joint is the glenoid fossa, located at the upper lateral aspect of the scapula. The scapula (the shoulder blade) lies on the posterior surface of the thoracic cage with its ventral surface concave, corresponding to the convex surface of the rib cage (Fig. 1–10). The only bony connection of the scapula with the thorax and vertebral column is through the acromioclavicular joint, with the clavicle attached to the sternum, which is attached to the vertebral column by way of the ribs (see Fig. 1–1).

The only other attachments of the scapula, hence of the total shoulder girdle, are by numerous muscles that are in turn attached to the vertebral column.

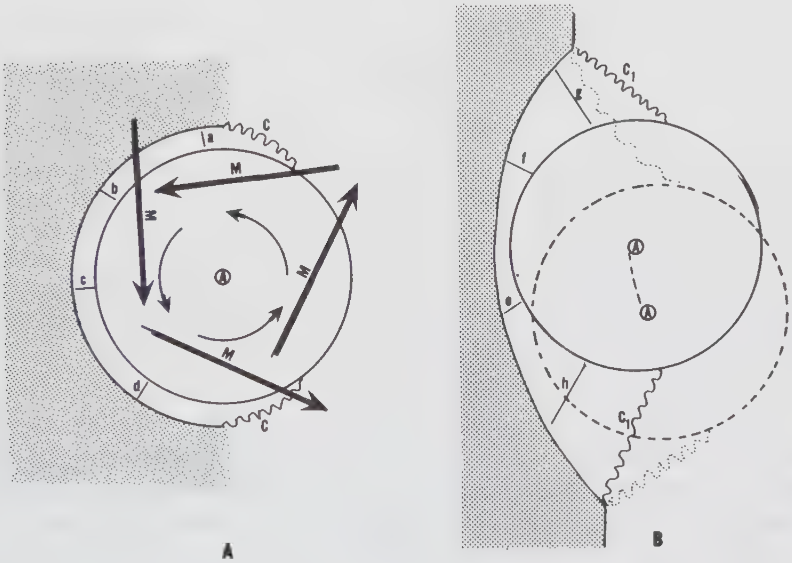


Figure 1–9. Congruous-incongruous joints. *(A)* In a congruous joint, the concave and convex surfaces are symmetrical. The articular surfaces are equidistant from each other at all points along their circumference (a = b = c = d, etc.). In rotation, movement occurs about a fixed axis (A). Muscular action (M) is that of symmetrical movement about this fixed axis and is needed for motion, not stability. The depth of the concave surface gives the joint stability. The capsule (C) has symmetrical elongation. *(B)* Incongruous joints have asymmetrical articulatory surfaces. The concave surface is elongated and the convex surface is more circular, thus the distance between them varies at each point (g>f>e<h). As the joint moves, the axis of rotation (A) shifts, and joint movement is that of gliding rather than rolling. Therefore, muscles must slide the joint and simultaneously maintain stability. The capsule varies in its elongation at all levels of movement. The glenohumeral joint is an incongruous joint.

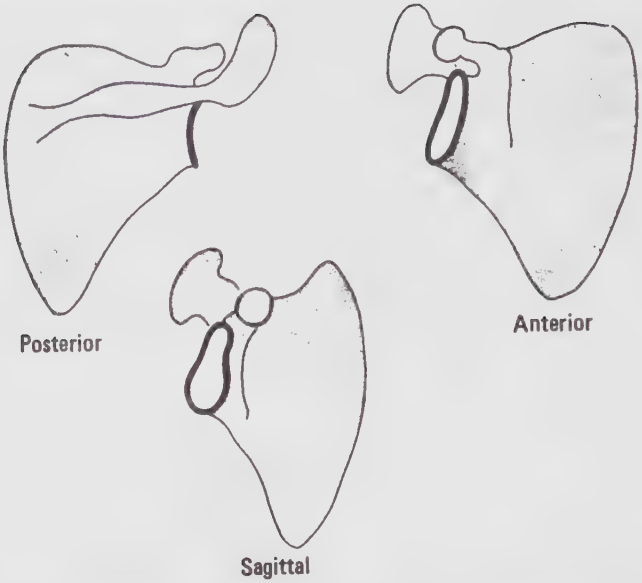


Figure 1–10. The scapula. The posterior, anterior, and sagittal views of the scapula are shown. The spine of the scapula seen on the posterior view divides the blade into the supraspinatus and infraspinatus fossae, from which originate the muscles that also bear these names. The sagittal view is more graphically seen in Figure 1–2, which depicts the relationship of the glenoid fossa to the overhanging acromial process and the medially located coracoid process. Note the angle of the glenoid fossa, facing laterally, anteriorly, and upward.

The dorsal aspect of the scapula is divided by a spinous bony ridge that extends horizontally from the inner (medial) aspect of the blade to extend laterally past the glenoid fossa as a bulbous enlargement termed the *acromial process*. The acromial process has a concave articular fossa in its anterolateral aspect into which is attached the end of the clavicle, forming the *acromioclavicular (A-C) joint*.

Above the spine of the scapula is a deep cavity which contains the supraspinous muscle belly. Below the spine is a hollow filled with the infraspinatus and teres minor muscle. These muscles form the bulk of the shoulder blade muscles as seen under the skin of the upper back. By their subcutaneous position they can be palpated to ascertain their bulk, revealing atrophy when it is present. These supraspinous and infraspinous muscles extend to join a tendon known as the *conjoined tendon*, the *supraspinatus tendon*, or *rotator cuff muscles tendon*.

On the inner surface of the scapula is a bony process that protrudes anteriorly and also ends in a bulbous aspect. This is the *coracoid process* and is located inward from the anterior margin of the glenoid fossa and anteroinferiorly from the acromial process. The coracoid process is palpable by an examiner in

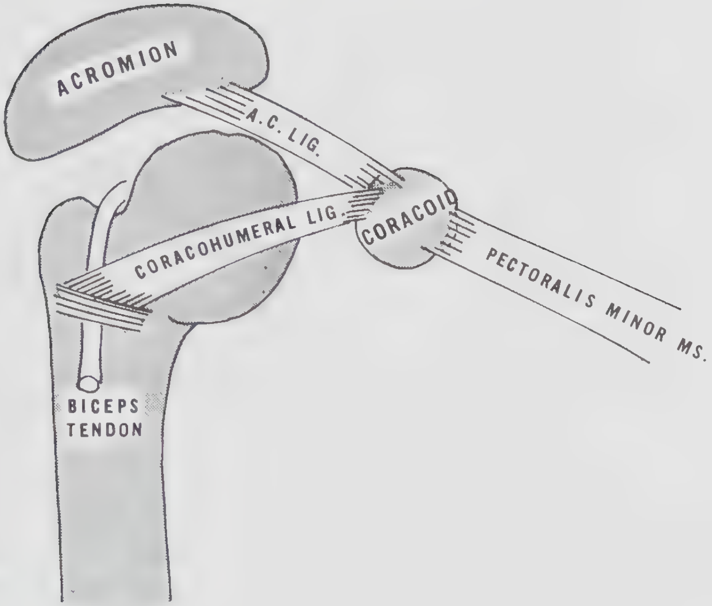


Figure 1–11. The coracohumeral ligament.

the region of the axilla under the lateral aspect of the clavicle.

The coracoid process is the point of attachment of scapular muscles such as pectoralis major and pectoralis minor. It also is the point of attachment of several ligaments—namely, the coracoclavicular, coracohumeral, and coracoacromial ligaments.

The coracoclavicular ligament functions upon the clavicle in scapular circumduction upon the thoracic wall, and it will be discussed in that capacity.

The coracohumeral ligament, attached from the coracoid process to the superior anterior aspect of the shaft of the humerus (Fig. 1–11), acts to limit external rotation of the arm.

The coracoacromial ligament is a broad ligament attached to both these bony prominences. Because it is fixed upon the scapula, the ligament functions merely as a roof above the glenohumeral joint (Fig. 1–12). The space between the coracoacromial ligament and the glenohumeral joint is termed the *suprahumeral joint* (see (2) in Fig. 1–1).

GLENOID FOSSA

The glenoid fossa is a shallow pear-shaped joint, with the narrow portion superiorly placed. Located on the anterior superior lateral aspect of the scapula (see

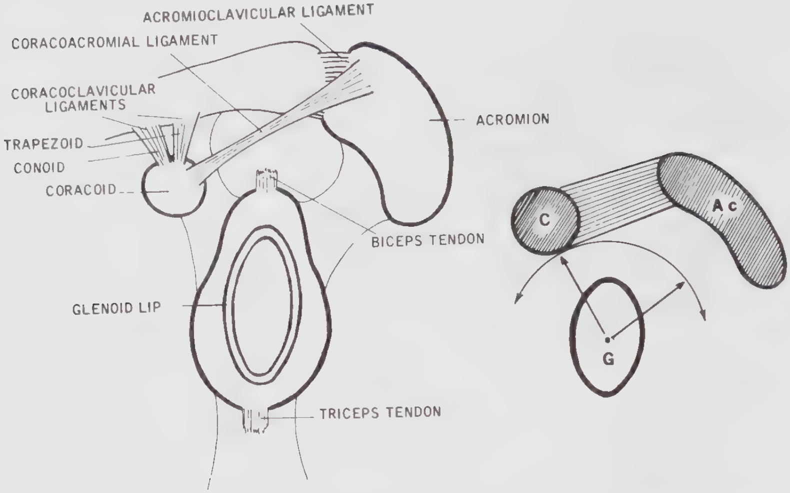


Figure 1–12. The acromiocoracoid arch. The diagram depicts the shape of the glenoid fossa and its relationship to the acromial process, the coracoid process, and the coracoacromial ligament. In essence this diagram shows the socket of the glenohumeral joint and also portrays the relationship of the suprahumeral joint.

Fig. 1–9), the glenoid fossa faces in the direction so placed by the position of the scapula. It normally faces forward, outward, and upward at a slight angulation.

It is the shallow portion of an incongruous joint and, thus, does not function to seat the head of the articulating humeral head. It is deepened by a fibrous labrum around its entire periphery. This labrum is considered to be a plication of the capsule of the glenohumeral joint which also attaches around the entire periphery. There is a firm fibrous attachment to the periosteum of the glenoid fossa, and this attachment presents a potential site of avulsion as a result of external trauma.

The base of the glenoid fossa is coated with cartilage, but because the head of the humerus is significantly more convex than the glenoid is concave, only a small portion of either cartilage surface is in contact at any time.

Motion between the shallow glenoid surface and the more convex surface of the humeral head is thus incongruous *gliding*.

GLENOHUMERAL CAPSULE

The capsule of the glenohumeral joint is very thin walled and spacious, holding as much as an estimated 30 cubic centimeters of fluid or air. At its attachment to the glenoid bone a small portion of the epiphyseal line extends into the capsular confines; this is why osteomyelitis can sometimes extend into this joint.

The capsule arises from the glenoid fossa (labrum) and inserts around the anatomic neck of the humerus. There is synovial lining throughout the capsule that blends with the hyaline cartilage of the head of the humerus. At the glenoid portion, however, it fails to reach the cartilage of the fossa (Fig. 1–13).

The long head of the biceps attaches to the superior aspect of the glenoid fossa (see Fig. 1–12). It invaginates the capsule but does not enter the synovial cavity. The biceps tendon is thus *intracapsular* but remains extrasynovial. The capsule folds and incorporates the biceps tendon down into the intertubular sulcus of the humerus and ends blindly at the site on the humerus opposite the insertion of the pectoralis major muscles (see Fig. 1–12).

With the arm hanging loosely at the side of the body, the upper portion of the capsule is taut and the inferior portion is redundant and pleated (Fig. 1–14). With the arm fully extended overhead the opposite exists: The inferior aspect of the capsule becomes taut, and the superior aspect becomes slack.

The superior portion of the capsule, taut when the arm is dependent, does not essentially support the arm and prevent downward subluxation. The capsule

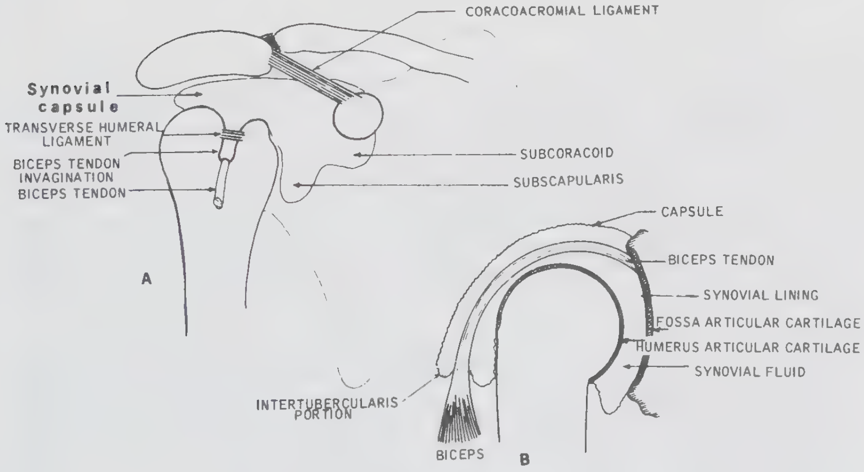

CORACOACROMIAL LIGAMENT
Synovial capsule
TRANSVERSE HUMERAL LIGAMENT
BICEPS TENDON INVAGINATION
BICEPS TENDON
SUBCORACOID
SUBSCAPULARIS
A
CAPSULE
BICEPS TENDON
SYNOVIAL LINING
FOSSA ARTICULAR CARTILAGE
HUMERUS ARTICULAR CARTILAGE
SYNOVIAL FLUID
INTERTUBERCULARIS PORTION
BICEPS
B

Figure 1–13. The glenohumeral synovial capsule. *(A)* The spacious capsule covers the entire humeral head. The invagination of the capsule accompanying the biceps tendon down the bicipital groove passes under the transverse humeral ligament at the level of the point of attachment of the pectoralis major muscle to the shaft of the humerus. The subscapularis and subcoracoid pouches of the capsule contain synovial fluid and are in direct continuity with the major capsule. These pouches are clearly seen in dye arthrograms. *(B)* The intracapsular, extrasynovial invagination of the long head of the biceps tendon as it proceeds to attach to the superior rim of the glenoid fossa. The synovial lining attaches to the articular cartilage of the head of the humerus but attaches to the glenoid fossa at a distance from the rim of the glenoid cartilage.

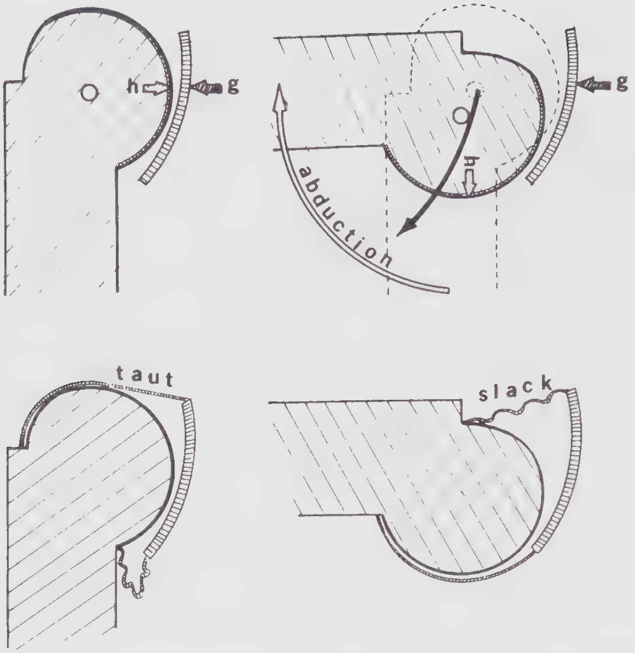


Figure 1–14. Capsular action during glenohumeral movement. The upper drawing depicts the *gliding* joint motion between the head of the humerus and the glenoid fossa. The arcs of both joint surfaces differ and thus form an incongruous joint surface relationship. The lower left drawing shows the arm dependent with the superior portion of the capsule taut, which prevents downward movement. The lower right drawing shows the arm abducted, which relaxes the superior portion of the capsule and causes the inferior portion to become taut. In the half-abducted arm both superior and inferior capsules are slack, which position is thus one of instability of the glenohumeral joint.

does not have the resilience to perform this function but assists the supraspinatus muscle, the function of which is to prevent downward movement of the dependent arm statically. When the supraspinatus muscle fails to support the arm, the capsule becomes involved but elongates and allows some downward subluxation.

Rotation of the arm at the glenohumeral joint involves the capsule in a similar manner. The posterior capsule tightens when the arm rotates internally, and the anterior capsule tightens when the arm rotates externally. It must be remembered that internal and external rotation of the head of the humerus upon the incongruous glenoid fossa causes anterior and posterior gliding as well as rotation around the horizontal axis.

The capsule is thickened into strands that essentially function as ligaments to restrict excessive external rotation (Fig. 1–15). This capsular restriction of rotation is illustrated in Figure 1–9.

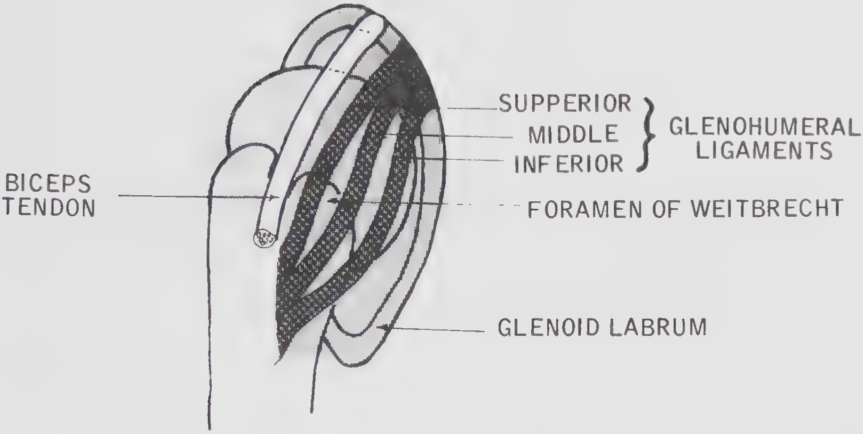


Figure 1–15. The anterior capsule and the glenohumeral ligaments. The glenohumeral ligaments that reinforce the anterior joint capsule are mere folds of the capsule. Three in number, they are fan-shaped, attach from the humerus, and converge toward the glenoid rim. The foramen of Weitbrecht may be covered by a thin layer or may be open as a communication between the joint space and the subscapular recess.

These glenohumeral ligaments are pleated folds of the anterior portion of the capsule, resulting in a fan shape with its base attached to the humerus. They reconverge to attach upon the anterior superior rim of the glenoid fossa and the adjacent bone.

There is a recess in the anterior capsule that forms a pouch. This is visualized upon arthrography and may suggest a tear, but this pouch is physiologic. The capsule is significantly loose, allowing the head of the humerus to be withdrawn from the fossa by as much as 3 cm. This is termed *joint play* (Fig. 1–16), and it is physiologic passive range of motion that cannot be actively elicited but must exist to permit full range of motion of an incongruent joint.

There is also an opening between the superior and middle ligamentous folds of the glenohumeral folds of the capsule (see Figure 1–15), termed the *foramen of Weitbrecht.* This foramen is covered merely by a thin layer of capsule and may be a communication between the capsular contents and the subscapular recess, but it does present a weak spot in the capsule that permits anteroinferior dislocation of the head of the humerus.

External rotation of the humerus must be physiologically limited to prevent subluxation, which occurs anteriorly and inferiorly through weak spots within the capsule. External rotation is limited by the glenohumeral ligaments of the capsule, the coracohumeral ligament, and the subscapularis muscle of the rotator cuff.

Constriction of the capsule also plays a major role in *frozen shoulder* attributed to *adhesive capsulitis.* This will be thoroughly discussed later.

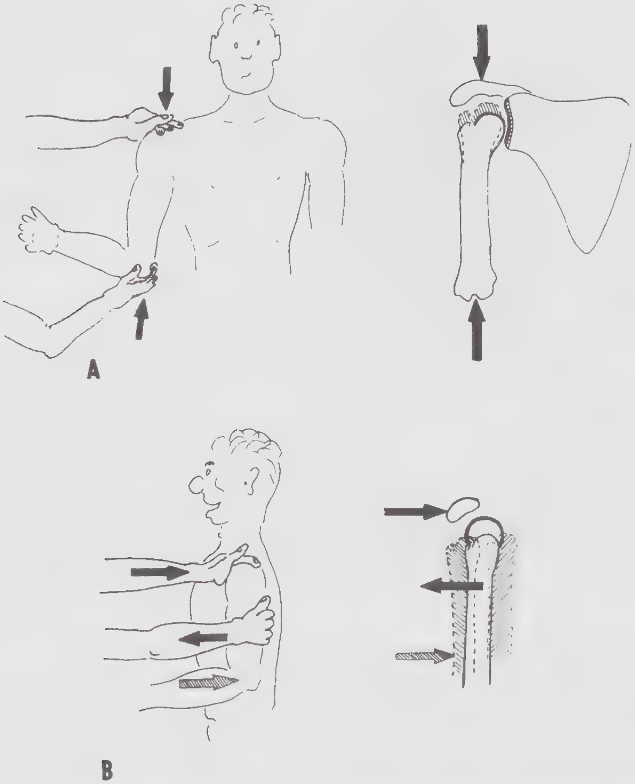


Figure 1–16. Manipulation treatment of *involuntary* motion of the glenohumeral joint. *(A)* Elevation of the head of the humerus against the glenoid fossa. Pressure along the shaft of the humerus, with the other hand preventing elevation of the scapula, causes the humerus to elevate, thus stretching the superior capsule. *(B)* Anterior and posterior motion of the head of the humerus against the glenoid. Three points of contact must be applied. One hand mobilizes the humerus while the other hand "fixes" the scapula. The elbow or forearm is fixed by the therapist's body or elbow.

THE SUPRAHUMERAL JOINT

The suprahumeral joint is not to be mistaken for a true joint, which is defined as an articulation; the point of juncture between two bones (Taber's). The suprahumeral joint is an articulation between the head of the humerus and the overlying coracoacromial ligament *and* the overhanging acromial process. The nearby presence of the acromium justifies the term *joint*, but there is no intervening cartilage, capsule, and synovium between these two articulating bones.

The overhanging arch protects the glenohumeral joint from direct overhead trauma. It prevents upward subluxation of the humerus, and it presents a

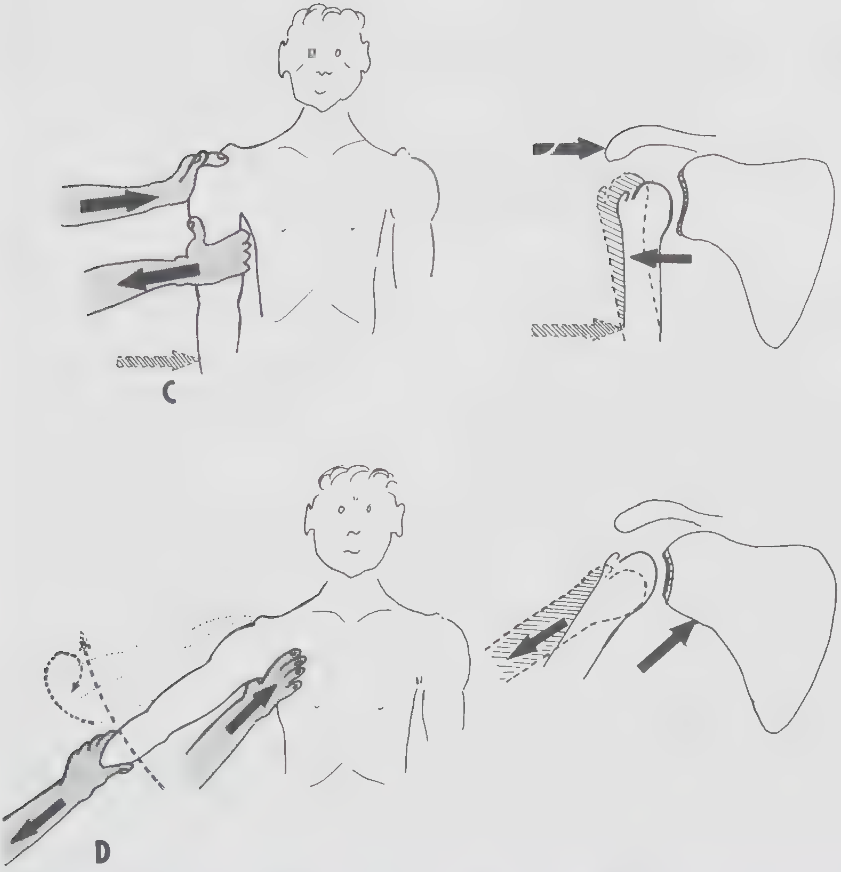


Figure 1–16 *(continued). (C)* Lateral motion of the head of the humerus away (in separation) from the glenoid. One hand of the therapist pulls at a right angle to the shaft of the humerus while the scapula and the elbow are *fixed. (D)* Traction to separate the head of the humerus from the glenoid while abducting and gradually externally rotating the arm. Counterresistance (fixation) is applied against the axillary border of the scapula.

mechanical obstruction to the humeral head as the arm performs abduction, forward and posterior flexion, and overhead elevation. This limitation constitutes the basis for some of the pathologic processes that occur at this joint.

The suprahumeral articulation is bounded within by the glenoid fossa and its labrum, superiorly and slightly anteriorly by the acromial process, medially by the coracoid process, and superiorly by the coracoacromial ligament (see Figure 1–12).

Within the contents of the suprahumeral joint space are found the subacromial bursae, the subcoracoid bursa, the supraspinatus muscle tendon, the superior aspect of the glenohumeral capsule, and a portion of the biceps tendon. Many of these tissues contained within a small space are supplied by nociceptor nerve endings capable of resulting in pain as well as in impairment.

MUSCULATURE OF THE GLENOHUMERAL JOINT

There are numerous muscles involved in glenohumeral joint function, both to support the upper extremity passively and to move it. These muscular functions are *static* and *kinetic*.

Five of the nine muscles related to the glenohumeral joint can be considered prime movers. They work in isolation and in concert and are delicately neurologically coordinated. Their neurologic control is conscious and unconscious, with an intrinsic feedback system of coordination.

In static support the muscles most significantly involved are the deltoid and the supraspinatus muscles. These two primarily maintain the head of the humerus up close to the overhanging coracoacromial arch and simultaneously keep the head of the humerus seated within the glenoid fossa. The other muscles of the rotator cuff, as these combined muscles are labeled, are the infraspinatus, teres minor, teres major, and the subscapularis. These muscles are ancillary to the function of stabilizing the head of the humerus as compared with the supraspinatus muscle and the deltoid.

The supraspinatus muscle originates from the supraspinatus fossa of the scapula above the spine of the scapula (Fig. 1–17). The muscle extends laterally, passing under the coracoacromial ligament ultimately to attach to a tendon on the greater tuberosity of the head of the humerus (Fig. 1–18). The greater tuberosity is located lateral to the bicipital groove. Its innervation is the suprascapular nerve, which is composed of cervical roots C_4, C_5, and C_6.

Responsible for supporting the weight of the upper extremity by sustained tonus, this muscle does so by intrafusal muscle spindle system and tendon Golgi innervation. Only when the muscle has been elongated past its normal length during sustained tone does the muscle fascia become the supporting tissue (Fig. 1–19).

All voluntary muscles in the body have a coordination control exerted by the spindle system: the intrinsic muscle fiber. The supraspinatus muscle is singled out at this juncture, but all muscles of the shoulder girdle complex are similarly innervated, ensuring a smooth, coordinated neuromuscular function.

MUSCLE SPINDLE SYSTEM

Every major muscle has extrafusal fibers that contract upon willful command through stimulation of the anterior horn cell within the gray matter of the

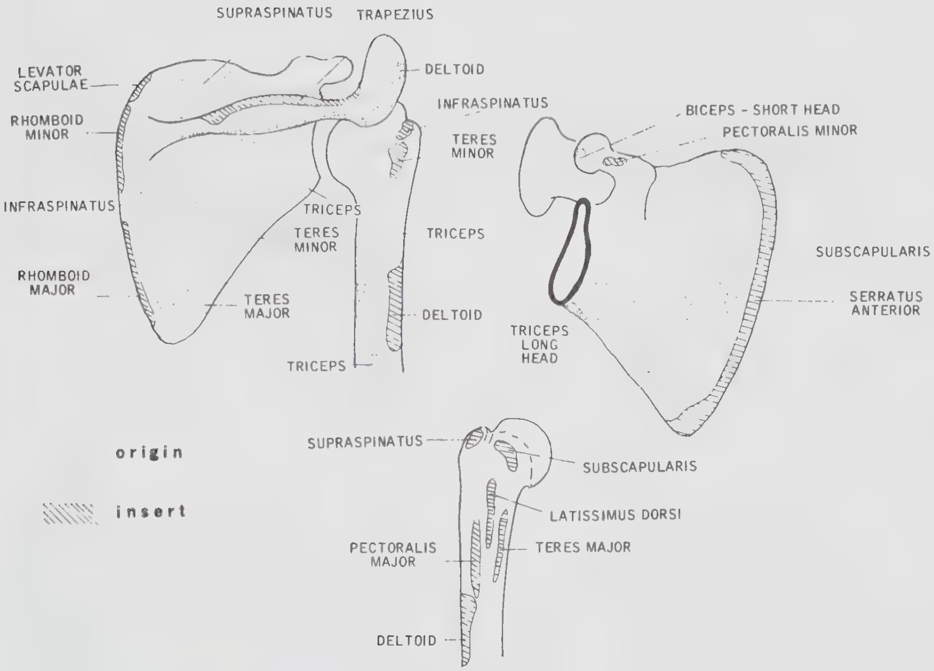

SUPRASPINATUS
TRAPEZIUS
LEVATOR SCAPULAE
DELTOID
INFRASPINATUS
RHOMBOID MINOR
TERES MINOR
BICEPS - SHORT HEAD
PECTORALIS MINOR
INFRASPINATUS
TRICEPS
TERES MINOR
TRICEPS
SUBSCAPULARIS
RHOMBOID MAJOR
TERES MAJOR
DELTOID
SERRATUS ANTERIOR
TRICEPS LONG HEAD
TRICEPS
SUPRASPINATUS
origin
SUBSCAPULARIS
▨ insert
LATISSIMUS DORSI
PECTORALIS MAJOR
TERES MAJOR
DELTOID

Figure 1–17. Sites of muscle origin and insertion upon the scapula and the humerus. All the muscles that perform shoulder girdle function are shown here. The stippled areas indicate the places from which the muscles originate, and the hatched areas represent spots upon which the muscles or their tendons insert.

cord. The fiber to the muscle, the alpha fiber, ends in a flowery neuromuscular aspect within the muscle.

The amount of tone and the speed of contraction required to perform the expected function is coordinated by the intrafusal spindle system. The strength of the contraction is also sensed and coordinated by the tendon organ (Golgi) system of that particular muscle.

In the mammalian muscle there are two types of intrafusal fibers in the intrafusal muscle spindle. The first type contains many nuclei in the central mass of the muscle fiber. This dilatation of the fiber is termed *nuclear bag*. The second type is termed a *nuclear chain fiber* and contains no dilated central bag (Fig. 1–20).

There are sensory nerve endings about these two spindle fibers that wrap around each fiber. These are termed *Ia* and *II*, with the Ia supplying the nuclear bag and the II the chain fiber. They project to the cord through the dorsal root ganglion to end in the gray matter of the cord. By way of internuncial fibers they cross over to synapse on the anterior horn cell (see Fig. 1–19).

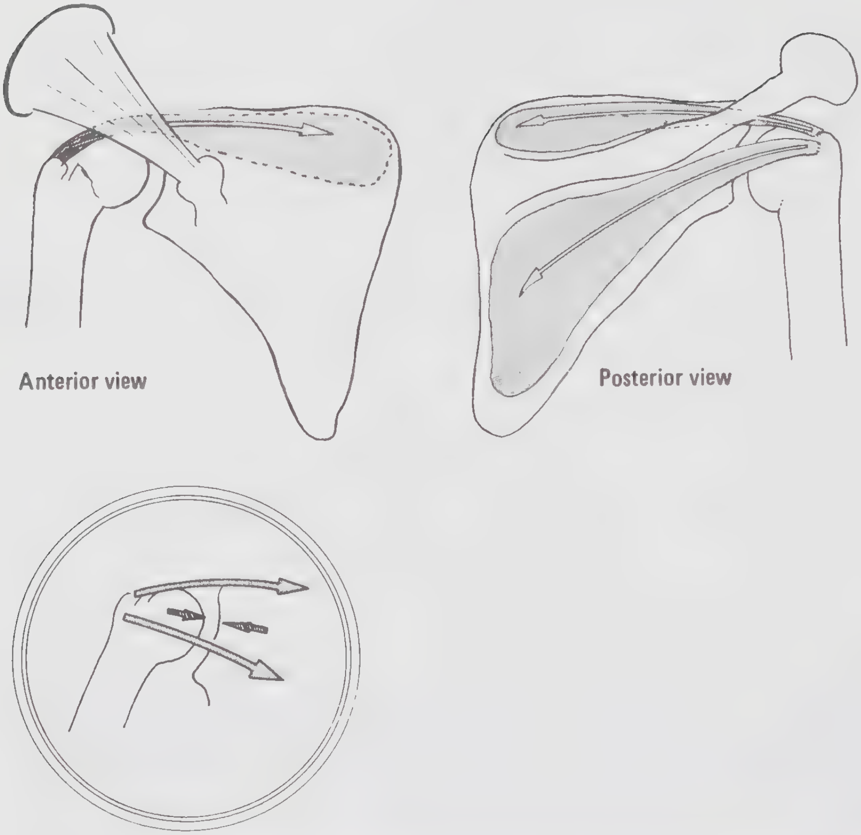


Figure 1–18. The supraspinatus muscle and the infraspinatus muscle. (*Anterior view*): The supraspinatus muscle originates from the supraspinatus fossa of the scapula and passes laterally under the coracohumeral ligament to attach upon the greater tuberosity of the humerus. (*Posterior view*): The infraspinatus muscle originates from the infraspinatus fossa and inserts upon the greater tuberosity just below the insertion of the supraspinatus tendon. The combined action of these two muscles (*insert*) brings the head of the humerus against the glenoid fossa in a slightly downward direction.

When the spindle cell is elongated, it sends messages to that effect to the cord by II sensory fibers. When the muscle is relaxed (thus shortened), the nuclear bag enlarges and thus also sends messages via sensory Ia fibers to the cord.

The spindle fibers also have a motor nerve supply which initiates the needed contraction to establish the length of the fiber needed for the projected function. As the muscle, extrafusal fibers, and thus intrafusal fibers, repeatedly lengthen and shorten in frequent activities, the *controlling* intrafusal fiber must be constantly and instantaneously *readjusted*.

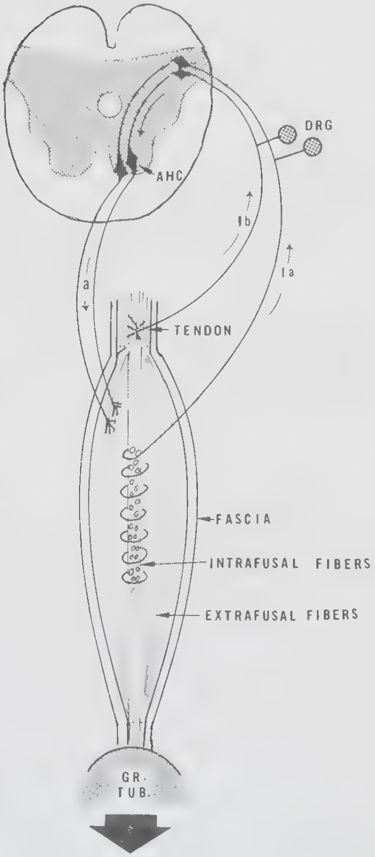


Figure 1–19. Spindle system function. The spindle system (intrafusal fibers) is parallel with the extrafusal fibers. When stretched, they signal the cord by way of Ia (from the spindle) and Ib (from the Golgi tendon organs) through the dorsal root ganglia (DRG). An interneural connection to the anterior horn cells (AHC) causes appropriate contraction of the extrafusal fibers.

The fascia elongates according to its physiologic limits. In this illustration the muscle is the supraspinatus attached to the greater tuberosity (Gr. Tub.) of the humerus.

These spindle motor nerves are termed *gamma efferent fibers* and constitute about 30 percent of the ventral motor nerve roots that go from the anterior horn cells to the extrafusal fibers. They terminate upon the spindle fibers and function in both static and dynamic manners.

When an extrafusal muscle fiber is elongated, the intrafusal spindle fiber is also elongated. An action potential is generated in this elongation which proceeds to the cord along the Ia and II afferent fibers. When the extrafusal fiber stops stretching, the intrafusal fiber potentials also stop firing. These intrafusal firings are essentially sensory in reporting the rapidity, frequency, duration, and extent of the lengthening.

The extent, rapidity, and force of the stretch of the intrafusal fiber is coded in the cord. By way of an internuncial fiber, the anterior horn cell is activated to release appropriate alpha fiber potential to cause the extrafusal fibers to contract.

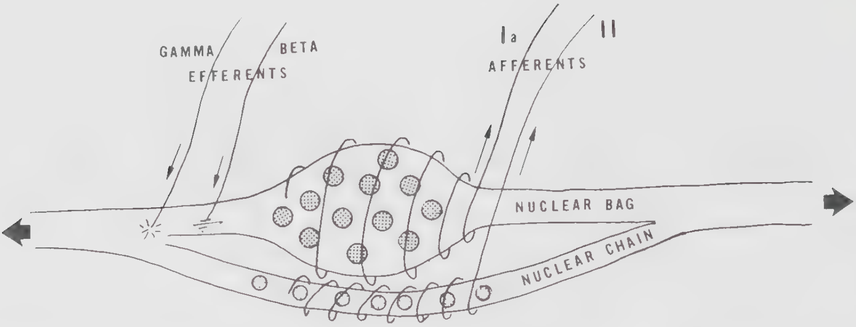


Figure 1–20. Intrafusal muscle spindle. The intrafusal spindle system has motor fibers through the gamma and beta efferents that control the length of the spindle. The sensory feedback from the spindle is transmitted by way of the Ia and II afferent fibers.

This is a *feedback system,* wherein the stretch of the intrafusal fibers (bag and chain) responds by sending a message to the cord, which in turn causes the extrafusal fibers to contract with appropriate force and rapidity.

Because the extrafusal fibers and thus the intrafusal fibers are constantly contracting and shortening as well as resting, the spindle system must be similarly lengthened and elongated. The spindle system is thus *set* for appropriate length and reaction. This is done reflexly by way of the gamma efferent motor fibers. It is apparent that this reflex feedback system ensures a smooth, coordinated, appropriate muscular response to the intended function.

There are many factors that influence the effectiveness of this system. Many remain unknown or suspected, but it is well accepted that fatigue plays an adverse effect upon the system, as does vibration. Anxiety causes an increased discharge of the gamma system, which possibly explains the increased tendon reflexes in anxious patients. It also partly explains the neurophysiologic basis of *tension myositis.* What other cortical and hypothalamic (limbic system) factors play a part remains conjectural.

THE ROTATOR CUFF

The supraspinatus muscle has already been discussed as being the principal muscle in statically supporting the humerus within the glenohumeral joint. The mechanical basis for supporting the head of the humerus within the glenoid fossa is based on the fact that the angle of the fossa forms an inclined plane. The head of the humerus, being a rounded structure, tends to roll downward and outward. The supraspinatus tendon attached to the head of the humerus prevents excessive rolling and thus prevents *subluxation* (Fig. 1–21).

It is also the principal kinetic muscle in initiating movement and function-

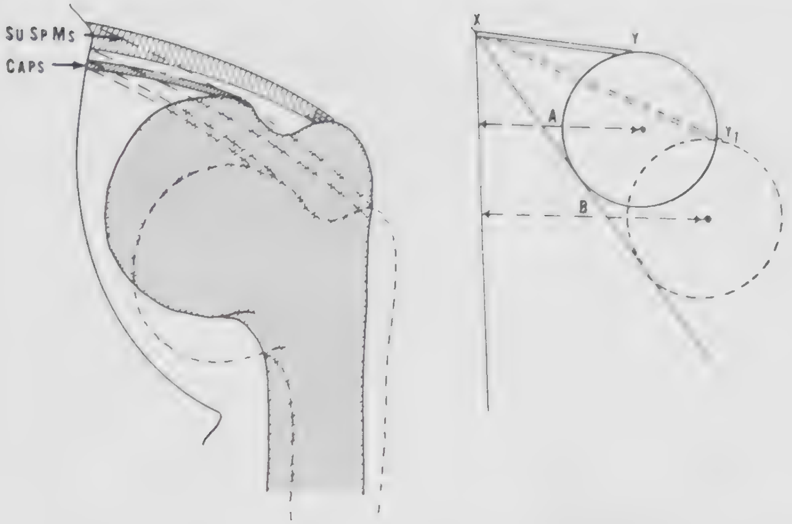


Figure 1–21. Capsular-passive cuff support. Due to the orientation of the glenoid fossa which faces forward, outward, and upward, the superior capsule—being taut in the normal position—becomes more taut as the humeral head A descends. B depicts an analogy of a ball rolling down an angled plane.

ally moving the arm in abduction, forward and posterior flexion, and external rotation within the glenoid fossa. It is the major component of the *rotator cuff*

Joining the supraspinatus muscle and forming the cuff is the *infraspinatus muscle.* This muscle originates from the greater surface area of the infraspinatus fossa of the scapula located immediately below the spine of the scapula (Fig. 1–18). It proceeds laterally to insert just below the attachment of the supraspinatus muscle tendon on the greater tuberosity. Its tendon and that of the supraspinatus muscle is also joined with the tendon to the teres minor muscle to form a *conjoined tendon*, or the *rotator cuff.* The infraspinatus muscle is innervated by the suprascapular nerve, having branches of roots C_4, C_5, and C_6.

The teres minor muscle arises from the lateral portion of the axillary border of the scapula (see Fig. 1–16) and passes laterally and upward to insert on the greater tuberosity of the humeral head immediately below the infraspinatus muscle tendon. The teres minor muscle is innervated by a branch of the axillary nerve as it proceeds to the deltoid muscle. It has C_5 and C_6 branches.

All three muscles—the supraspinatus, infraspinatus, and teres minor—end in a conjoined tendon (Fig. 1–22) with the tendon of the subscapularis muscle.

The subscapularis muscle (Fig. 1–23) is also considered to be a rotator cuff muscle. Its function differs from the other three cuff muscles and will be discussed at a later time. It originates from the entire anterior (thoracic) surface of

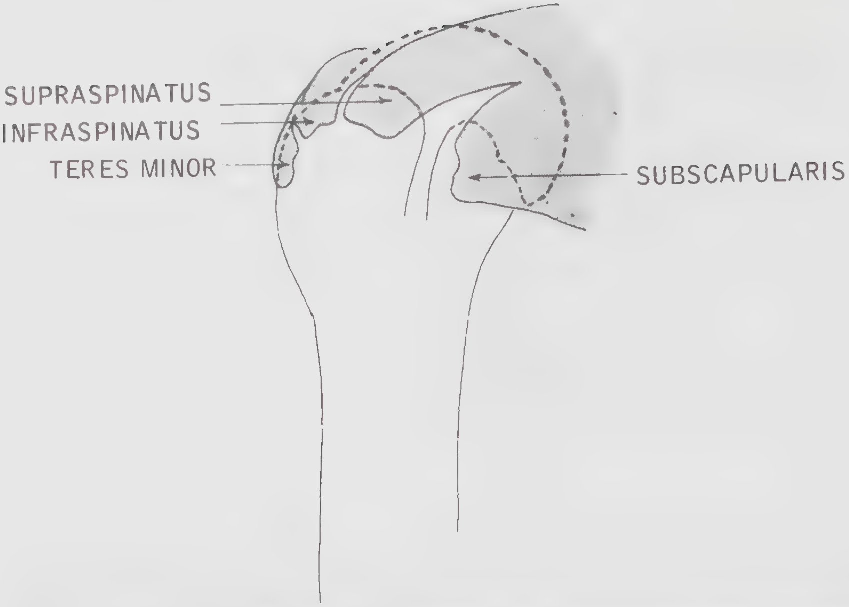


Figure 1–22. Rotator cuff insertion upon the humerus. The conjoined tendinous insertion of the four rotator muscles that comprise the cuff is viewed anteriorly. The supraspinatus attaches to the greater tuberosity; the infraspinatus, immediately below it; and then the teres minor. The subscapularis inserts upon the lesser tuberosity below the cartilage of the head and medial to the bicipital groove. There is a sulcus through which the biceps tendon emerges.

the scapula and proceeds laterally to attach to the lesser tuberosity of the head of the humerus. It is the most anterior and most medial muscle of the rotator cuff. It is innervated by the upper and lower subscapular nerve, with branches of C_5 and C_6 roots.

The lesser tuberosity is located just medial to the bicipital groove (see Fig. 1–22). The tendon of the subscapular muscle passes in front of the glenohumeral joint and is separated from the neck of the scapula by a bursa. This bursa is a pouching of the synovial joint of the glenohumeral joint (see Fig. 1–13).

There is an opening in the anterior portion of the cuff insertion upon the humeral head between the supraspinatus and subscapular muscles through which the biceps tendon (long head), its sheath, and an invagination of the synovial cavity pass. This opening is reinforced by the coracohumeral ligament which proceeds from the coracoid process and fuses with the anterior superior aspect of the glenohumeral capsule (see Fig. 1–2). There is further reinforcement from the *transverse humeral ligament* (see Fig. 1–13), which holds the biceps tendon in the bicipital groove.

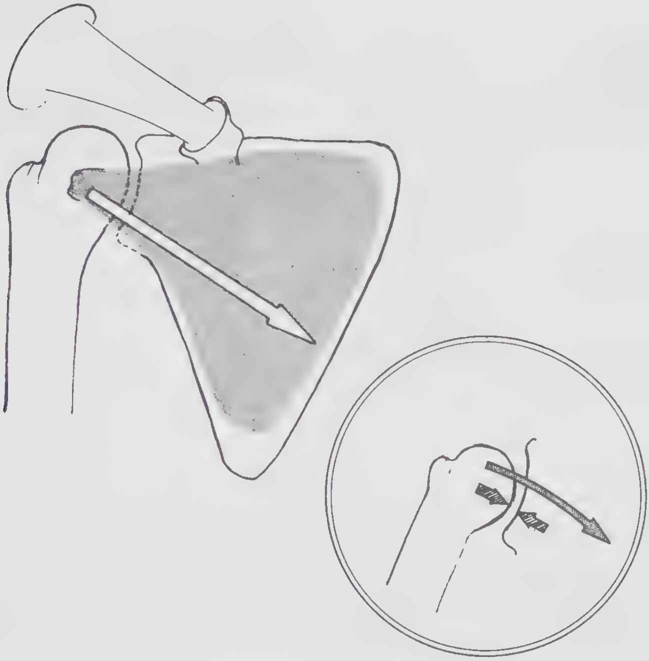


Figure 1–23. The subscapularis muscle. The subscapularis muscle originates from the entire anterior surface of the scapula (see Fig. 1–17), the surface that glides against the thoracic wall. The muscle extends laterally and attaches to the lesser tuberosity of the humerus. Its tendon is the most medial of those forming the cuff. Its action is to pull the head of the humerus into the glenoid fossa and slightly downward.

The Rotator Cuff Tendon

Tears that occur within the rotator cuff tendon are usually preceded by degenerative changes as well as by overwhelming traction or compressive forces. These tears usually occur longitudinally in the anterior portion of the cuff between the supraspinatus tendon and the coracohumeral ligament at the *critical zone.*

This so-called critical zone (Fig. 1–24) is the site within the conjoined tendon where degenerative changes and tearing occur. It was called critical because it was originally considered to be the region of vascular ischemia of the tendons. More recent studies have refuted the lack of adequate circulation; rather, the critical area has a profuse collateral anastomosis from branches of the anterior circumflex artery and the suprascapular and subscapular arteries. The former is the bony branch and the latter two muscular branches.

The critical zone varies from being ischemic when the anastomosis is constricted and hyperemic when it is allowed to flow freely. In the dependent arm,

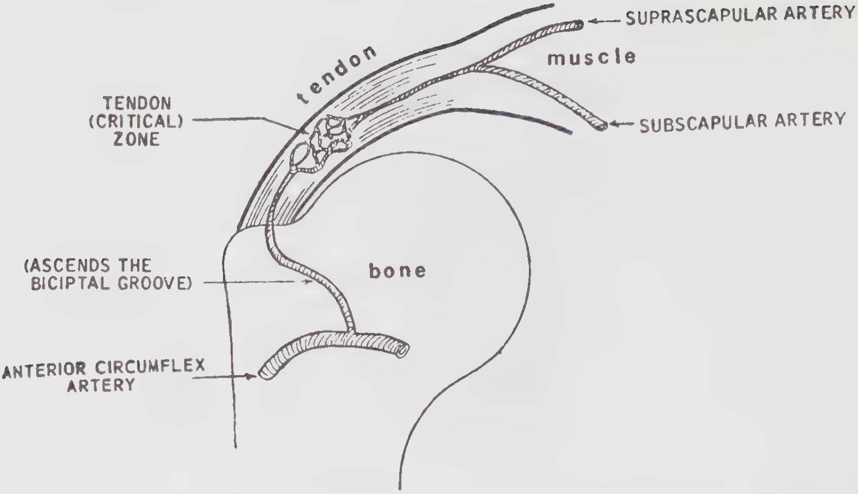


Figure 1–24. Circulation of the tendons of the cuff: the *critical zone*. The tendons of the cuff have a highly vascular zone at the anastomosis of the muscular vessels and the osseous vessels. This critical zone is the portion with the greatest tensile strength and is also the area that accumulates the calcium deposits; thus it is the site of cuff ruptures. This zone is graphically shown, and the contributing vessels are identified.

which elongates and thus compresses the arterial flow, the area is ischemic (Fig. 1–25). In the elevated, abducted, forward flexed arm when the rotator cuff muscles contract, the anastomosis is compressed, hence the area is ischemic. Only when the arm is passively supported and the rotator muscles are not contracted is the area hyperemic.

It is apparent that the area varies daily from ischemic to hyperemic, dependent upon nondependent rest or passive support of the dependent arm or active contraction during motion.

THE DELTOID MUSCLE

The *deltoid muscle* plays a significant role in the support of the dependent arm and in the kinetics of arm motion: abduction, forward and posterior flexion. The muscle arises anteriorly from the clavicle, laterally from the acromium, and posteriorly from the spine of the scapula. It has a broad base of origin. It ends in a tendon that passes down in front of, lateral to, and behind the glenohumeral joint to attach to the anterior lateral aspect of the middle third of the humerus (Fig. 1–26). The muscle is innervated by the axillary nerve (C_5 and C_6 roots).

Because of its origin and the fact that insertion is essentially in a vertical plane, the basic action of the deltoid muscle is to elevate the humerus up into

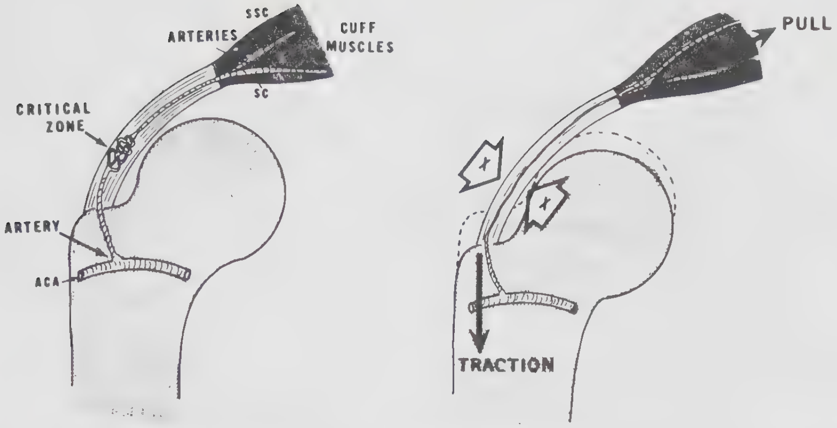


Figure 1–25. Blood circulation. *(Left)* Circulation to the rotator cuff. The arterial branch from the anterior circumflex artery (ACA) enters from the bone. The suprascapular (SSC) and the subscapular (SC) branches merge to enter from the muscle. The critical zone of the tendon is an anastomosis which is patent when the arm is supported and inactive. *(Right)* Traction upon the cuff from the dependent arm or from pull of the contracting cuff muscle elongates the tendon and renders the critical zone *(arrows)* relatively ischemic.

the overhanging coracoacromial ligament (see insert in Fig. 1–27). When working in harmony with the rotator cuff muscles, the middle fibers abduct the arm a slight degree from the midline of total dependency, that is, by contraction of the supraspinatus muscle. The anterior fibers flex the humerus forward in the sagittal plane while also slightly rotating the upper arm internally. The posterior fibers extend (posterior flex) the humerus in the sagittal plane while slightly rotating the humerus externally.

GLENOHUMERAL MOVEMENT

Motion of the glenohumeral joint (Fig. 1–27) is performed as a complex neuromuscular action with the articular motions permitted in an incongruous joint. In review, the head of the humerus is a rounded convex articular surface *gliding* about a large arc of the shallow concave glenoid fossa. As the arm abducts, the head of the humerus rotates about a descending center of rotation and glides downward upon the glenoid fossa. The fossa, and therefore the scapula, remains in a fixed position as a base of support.

The muscles of the scapula remain initially as isotonically contracting muscles to stabilize the scapula upon the thoracic cage. The coordinated muscular and articular action of the glenohumeral joint with simultaneous scapulotho-

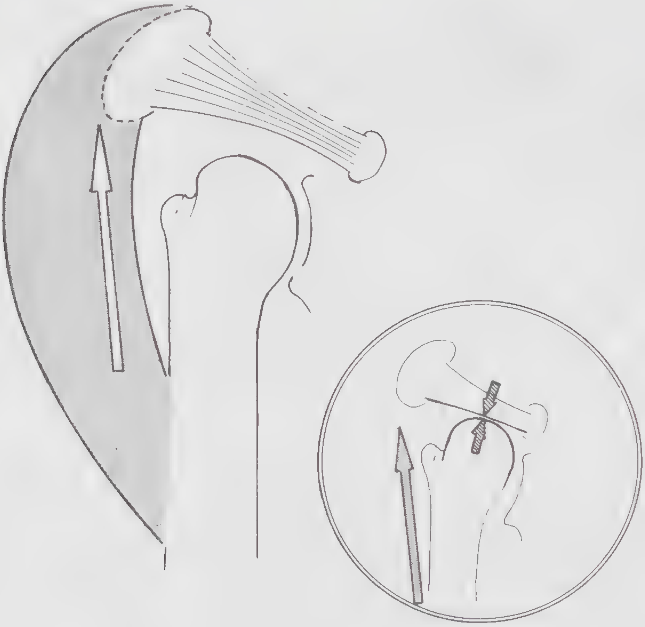


Figure 1–26. The deltoid muscle and its isolated function. The deltoid muscle originates from the inferior aspect of the spine of the scapula and the protruding acromial process (see Fig. 1–17). By its attachment into the humeral shaft, it has a direction of pull as depicted by the arrow in the large figure. Its isolated action shown in the circle is that of elevation, impinging the head of the humerus directly up under the coracoacromial arch. As the head of the humerus is rotated, depressed, and adducted into the glenoid fossa by the other cuff muscles, the deltoid becomes a powerful abductor.

racic motion is termed *scapulohumeral rhythm.*

The deltoid muscle, structurally adapted as a prime abductor, cannot act alone to abduct the humerus. Its line of action is saggital to the humerus, elevating the humerus with no abduction.

The rotator cuff, especially the supraspinatus muscle, inserts eccentrically into the axis of rotation of the head of the humerus. Its contraction thus approximates the head of the humerus into the glenoid fossa; it *seats* the joint. By its site of insertion, contraction of the supraspinatus begins abduction of the humerus (Fig. 1–28).

To abduct the arm, the deltoid muscle requires the rotator cuff (Fig. 1–29) to create an angle of abduction that allows the deltoid to act at an angle to the humerus. (Fig. 1–30).

As the arm abducts, the head of the humerus, gliding downward as well as rotating, gradually impinges upon the coracoacromial ligament and the overhanging acromium (see Fig. 1–28). The greater tuberosity, by virtue of its pro-

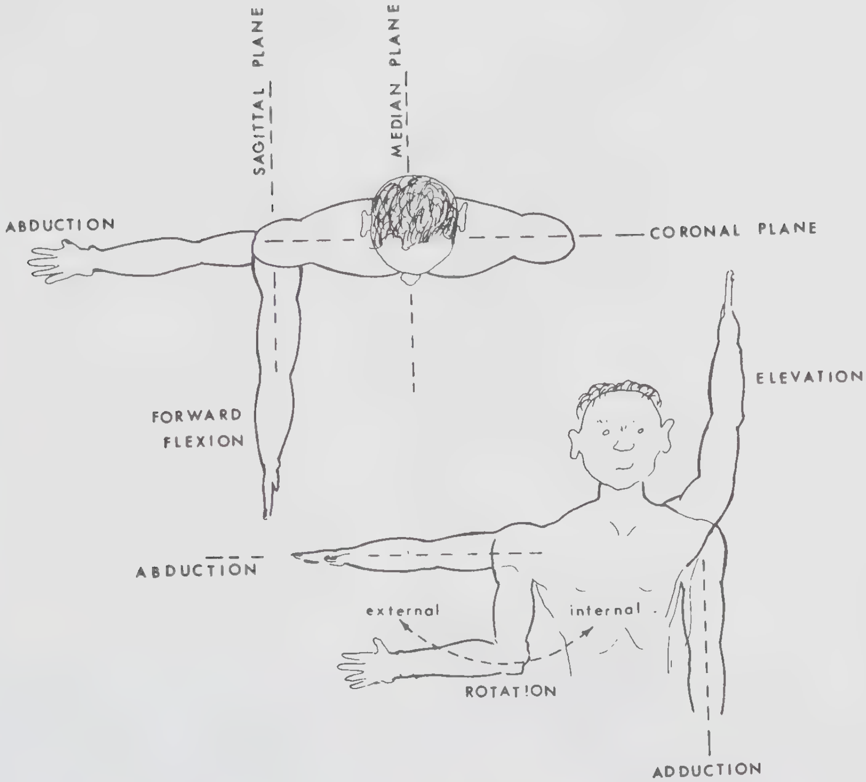
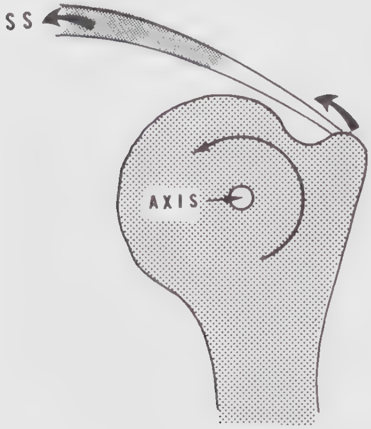


Figure 1–27. The planes of arm movement, indicating the direction of movement and the planes of movement in relation to the body. The body is viewed from above and from the front. All arm planes are related to these two body positions.

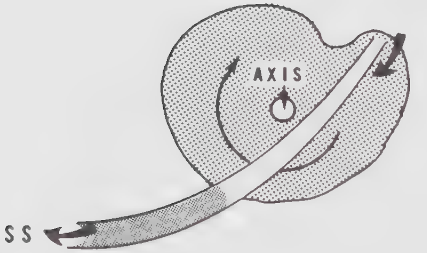
trusion past the outer surface of the head, impinges first upon the coracoacromial ligament.

The head of the humerus rotates and glides downward upon the glenoid fossa by the muscular action of the supraspinatus muscle, but this is in concert with the remaining muscles of the rotator cuff: the infraspinatus and teres minor muscles (Fig. 1–31). The infraspinatus muscle adducts (seats) the head of the humerus and rotates it about the axis. The teres minor muscle also rotates and seats the humeral head but, by pulling it downward, accentuates the downward glide action.

Early kinesiologic studies of the glenohumeral musculature maintained that the supraspinatus muscle initiated and acted during the first degrees of abduction, with its maximum action exerted at 100° of abduction. This has been disproven by Inman and colleagues, who have demonstrated that the supraspinatus muscle "acts during the entire abduction of the arm in the coronal plane." It



ANTERIOR VIEW



SUPERIOR VIEW

Figure 1–28. Function of the supraspinatus muscle. The anterior view shows supraspinatus function abducting the arm in the coronal plane. Viewed superiorly, this muscle externally rotates the arm. SS = supraspinatus muscle.

varies only quantitatively, depending on the force and speed of contraction needed during the total arm action and on the expected arm-head activities.

In the coronal plane, abduction of the humerus is limited by impingement of the greater tuberosity of the humerus upon the overhanging acromium and coracoacrominal ligament. This mechanical impingement occurs at 90° of abduction with the humerus (the upper arm) facing in the sagittal plane (see Fig. 1–27).

If the arm is rotated internally, the greater tuberosity is also rotated internally and impinges upon the coracoacromial ligament sooner, thus limiting abduction to approximately 60°. If the humerus is rotated externally, the greater tuberosity passes behind the overhanging acromium and coracoacromial ligament and allows greater abduction—to 120°, an increase of 30° from neutral, 60° from the internally rotated humerus.

It becomes apparent that overhead elevation of the arm, from neutral dependency to 120° of overhead elevation, requires abduction of the humerus with simultaneous external rotation.

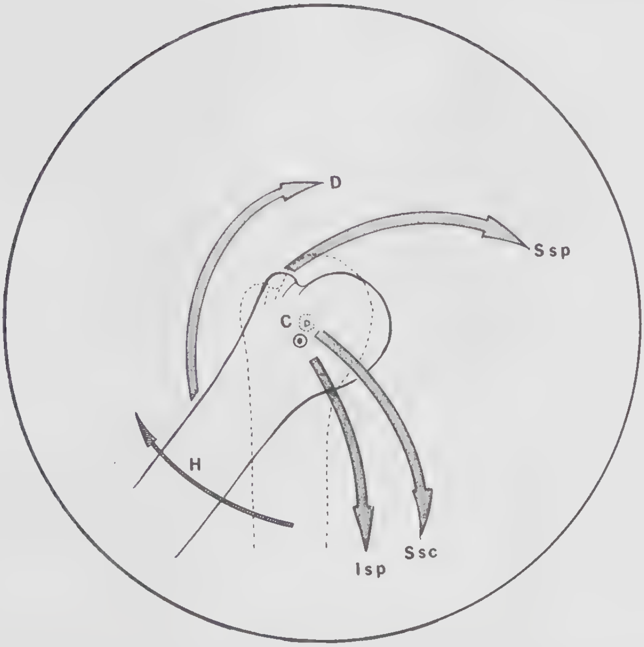


Figure 1–29. Combined cuff and deltoid action upon the glenohumeral articulation. Abduction of the humerus along the plane H is the result of combined action of the supraspinatus (Ssp) adducting the head into the fossa; the infraspinatus and subscapularis (Isp and Ssc) adducting and depressing the head; and the deltoid (D) acting as an abductor when working with these cuff muscles. The center of rotation (C) lowers during this downward gliding motion.

The rotation in the coronal plane has been demonstrated to be the combined function of the rotator cuff and deltoid muscle. The external rotation of the humerus during this abduction is also performed by the rotator cuff acting eccentrically about the axis of rotation of the shaft of the humerus (see lower figure in Fig. 1–28 and Fig. 1–32).

To summarize the humeral phase of the scapulohumeral rhythm:

1. Isotonic (initially) stabilizing contraction of the scapular muscles upon the thorax,
2. Isometric contraction of the rotator cuff muscles to initiate abduction of the arm,
3. Kinetic contraction of the deltoid muscle from the isotonic stabilizing function to become an abductor, and
4. Simultaneous isometric contraction of the rotator cuff to rotate the humerus externally as it abducts from 60° to 120° during abduction above the horizontal level.

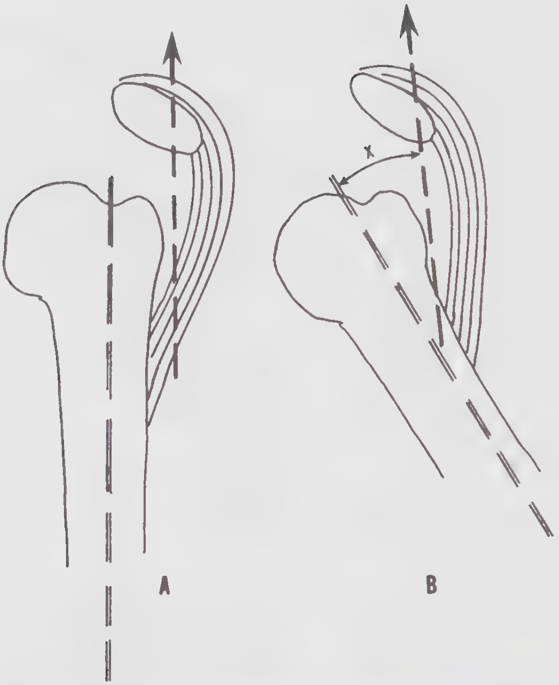


Figure 1–30. Abduction angle of deltoid. *(A)* With the arm dependent, the deltoid line of pull is along the line of the humerus and thus elevates the arm up against the acromion. *(B)* With slight abduction of the humerus the angle of the deltoid changes its pull to abduction of the arm.

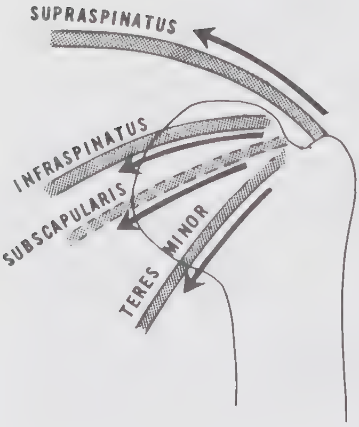


Figure 1–31. Rotator cuff mechanism. The supraspinatus muscle pulls the head of the humerus into the glenoid and slightly rotates the humerus into abduction. The infraspinatus muscle also rotates the head and pulls it slightly down. The teres minor muscle pulls in a more downward direction. The subscapular (subscapularis) muscle pulls the head into the glenoid, but its main rotatory action is to internally rotate the humerus about its longitudinal axis.

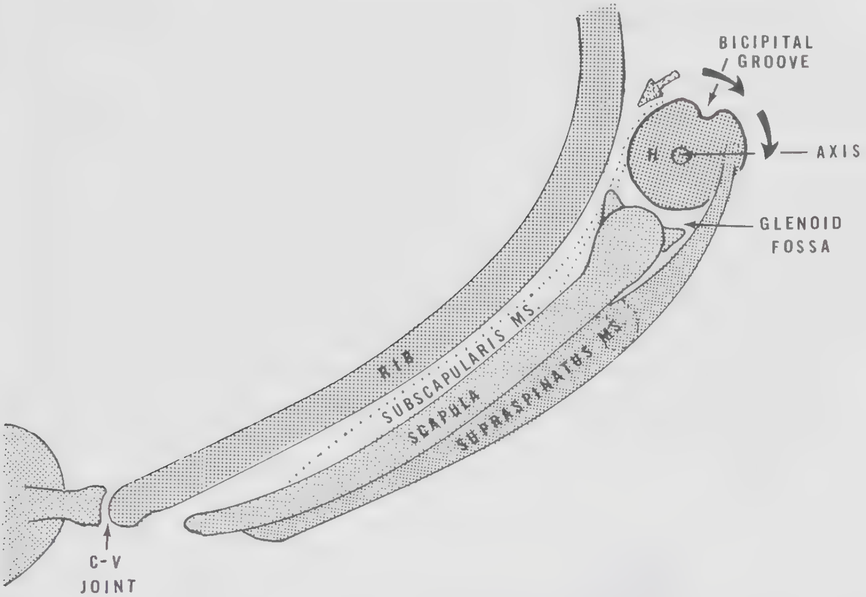


Figure 1–32. Supraspinatus external rotation of the humerus. Superior view of the scapulocostal joint depicting the supraspinatus muscle that attaches to the greater tuberosity located lateral to the bicipital groove. Being lateral to the axis of rotation, the attachment permits the supraspinatus muscle to rotate the humerus (H) externally. Located under the scapula, the subscapularis *(dotted)* attaches to the lesser tuberosity (medial to the bicipital groove) and thus is an internal rotator *(dotted arrow)* of the humerus. The ribs attach to the transverse processes of the thoracic vertebrae at the costovertebral (C-V) joints.

The subscapularis muscle, an abductor muscle upon the glenohumeral joint, is an internal rotator and thus must be inhibited (reciprocally relaxed) during this overhead elevation.

All these neuromuscular actions are dependent for their synchronization upon the feedback from the spindle and Golgi systems and the proprioceptive feedback from the periarticular tissues.

SCAPULAR COMPONENT OF THE SCAPULOHUMERAL RHYTHM

The scapula, which supports the entire upper extremity upon the thorax, also participates in a coordinated manner in any upper extremity function.

The scapula is a broad curved bone (see Fig. 1–32) that moves in a gliding manner upon the thoracic wall at what is termed the *thoracoscapular articulation.* Besides this articulation, the scapula is also attached to the upper trunk area by the clavicle at the *acromioclavicular joint* (A-C).

There are numerous muscles attached to the scapula that suspend and move the scapula. These are the trapezius, the serratus anterior, the rhomboids, levator scapulae, and the pectoralis major. By virtue of being attached to the upper humerus the latissimus dorsi also indirectly influences motion of the scapula.

In the motion of the arm—abduction, external rotation, forward and posterior flexion, and ultimate overhead motion—the major muscular function resides in the trapezius and serratus anterior muscles.

The broad fan-shaped *trapezius muscle* acts as three muscles (Fig. 1–33) in spite of a single innervation: the spinal accessory nerve (XI). The upper fibers

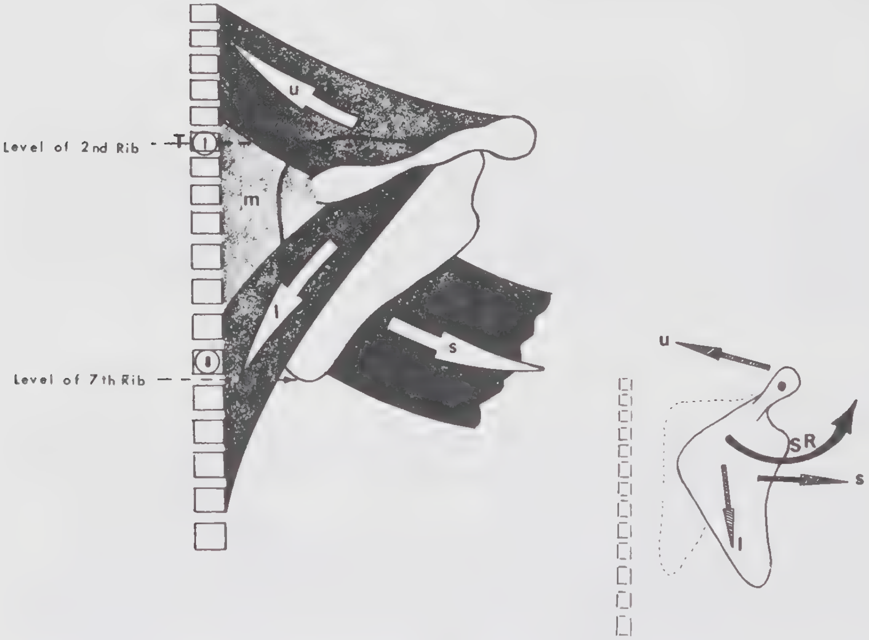


Figure 1–33. Scapular musculature: rotators. The scapular muscles forming the rotator phase of the scapulohumeral rhythm are shown with the upper trapezius fibers elevating the outer border of the spine, the lower fibers of the trapezius depressing the medial border of the spine, and the serratus pulling the lower portion of the scapula forward from its position under the blade. The combined action moves the scapula in orbit around the acromioclavicular center of rotation (u, m, and l = upper, middle, and lower trapezius; s = serratus anterior; SR = scapular rotation).

of the trapezius muscle originate from the ligamentum nuchae of the lower cervical spine, the posterior spinous processes of the cervical spine, and from some of the upper thoracic vertebrae. These fibers radiate laterally and downward to attach to the upper margin of the spine of the scapula. The action of these fibers is to pull the scapula upward with some inward rotation about the axis of the acromioclavicular joint (see insert Fig. 1–34).

The middle fibers originate from the spinous processes of the upper thoracic vertebrae and proceed laterally (horizontally) to attach to the medial border of the spine of the scapula. These middle fibers functionally *fix* the scapula during abduction of the arm. They reciprocally relax during forward flexion of the shoulder in the sagittal plane as the shoulder girdle moves en masse upon the costal wall.

The lower trapezius fibers of the trapezius muscle originate from the spinous processes of the lower thoracic vertebrae and attach to the inferior medial border of the spine of the scapula. These fibers pull the scapula down and

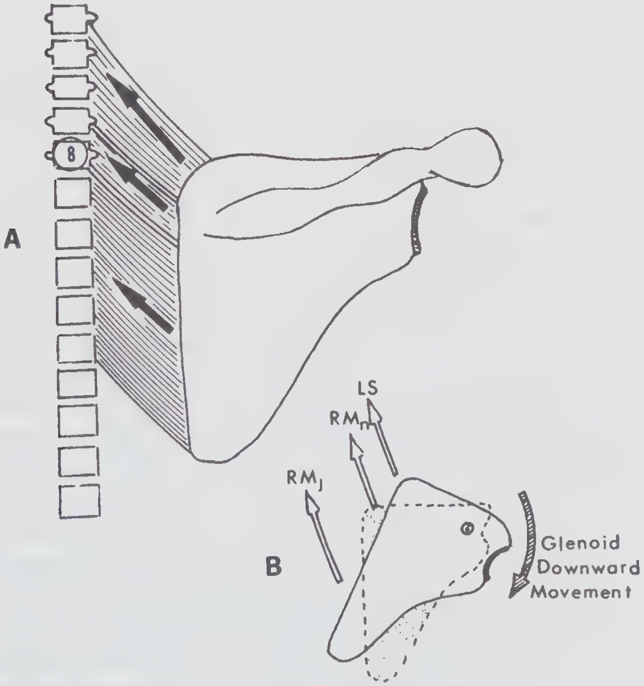


Figure 1–34. *(A)* Downward rotators of the scapula. *(B)* The muscles acting upon the scapula directly to cause downward rotation of the glenoid fossa are the levator scapulae *(upper arrow)*, the rhomboid minor *(middle arrow)*, and the rhomboid major *(lower arrow)*.

rotate the inferior angle of the scapula laterally away from the vertebral column.

All these trapezius functions move the scapula and thus also alter the facing direction of the glenoid fossa. In lateral rotatory motion from contraction of the trapezius muscle and glenoid fossa faces further up and forward. As the trapezius muscle relaxes, the glenoid fossa descends to its normal passive facing: forward, upward, and outward, albeit at a different angle.

The *serratus anterior muscle* is the other major muscle. Its function is forward flexion and upward rotation of the scapula during upper extremity action. This muscle is also broad, originating from the upper eight ribs of the anterolateral chest wall anterior to the scapula and proceeding posteriorly to insert into the medial vertebral border of the scapula. By its origin and insertion it occupies the space between the chest wall and the concave border of the scapula—the so-called scapulocostal joint.

From its origin and insertion it moves the scapula forward, but because of the motion about the acromioclavicular joint it also rotates the scapula about this axis and upward.

The serratus anterior muscle is innervated by the long thoracic nerve formed by branches of C_5, C_6, and C_7—primarily C_6—before these nerve roots enter the brachial plexus.

In summary, the combined action of the trapezius muscle bands and the serratus muscle is to move the scapula forward and to rotate it upward about the axis of the acromioclavicular joint on the rib cage. As the scapula moves, so moves the facing of the glenoid fossa.

Of the other muscles acting upon the scapula—the rhomboids, the levator scapula, and the pectoralis major—their action is contrary to the trapezius and serratus, and they must relax reciprocally when these two muscles (trapezius and serratus) contract (Fig. 1–34).

The minor rhomboid muscle originates in the lower cervical vertebrae immediately below the site of origin of the levator scapula muscle and proceeds laterally and downward to attach onto the vertebral border of the scapula. The major rhomboid muscle originates below the minor from the thoracic vertebrae and also proceeds laterally and downward to attach to the lower portion of the vertebral border of the scapula below the attachment of the minor. Both rhomboid muscles receive nerve supply from the dorsal scapular nerve.

Because of their angulation, contraction of the rhomboid muscles adducts the lower portion of the scapula and rotates the scapula downward about the axis of the acromiclavicular joint (see insert in Fig. 1–35).

The *levator scapula muscle* originates in the lower cervical vertebral transverse processes and proceeds laterally and downward to attach to the superior medial angle of the scapula (see Fig. 1–35). Its action is elevation of the scapula and rotation downward about the axis of the A-C joint.

Indirectly the latissimus dorsi muscle also affects the motion of the scapula. It originates in the spinous processes of the thoracic spine from T_6 down, then proceeds to the lumbar vertebrae and laterally to the medial aspect of the crest

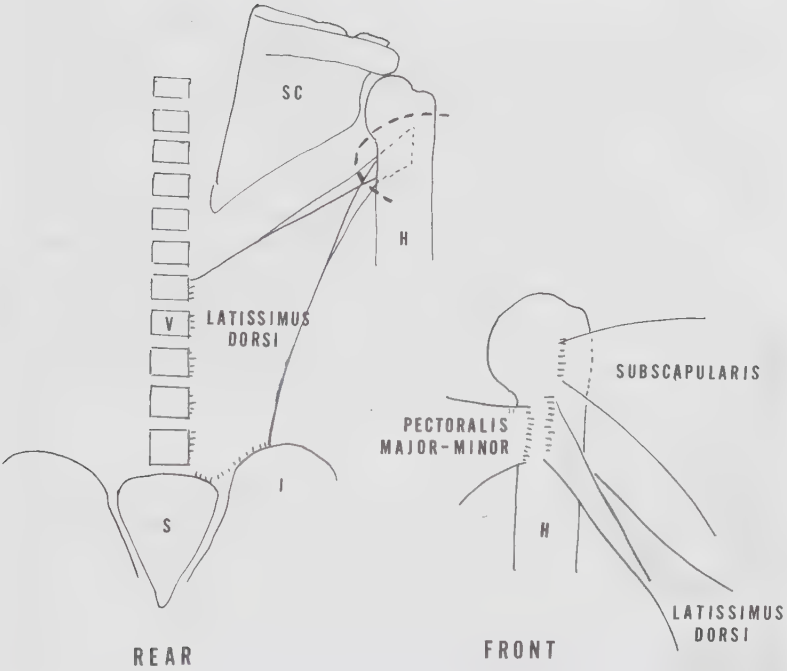


Figure 1–35. Latissimus dorsi and pectoralis muscle attachment upon the humerus.

of the ilium (Fig. 1–35). It inserts into the anteromedial aspect of the humerus upon the lesser tuberosity. Its function in relation to humerus is internal rotation and adduction of the arm at the glenohumeral joint, but it also depresses the outer angle of the scapula in conjunction with the rhomboid and the levator scapula muscles. The latissimus dorsi muscle is innervated by the medial and lateral thoracic nerves.

The *pectoralis major* muscle also affects the position and motion of the scapula. This muscle has an extensive origin: from the sternal half of the clavicle, the sternum, the costal cartilages of the second to the seventh ribs, and the fascia of the upper abdominal muscles (Fig. 1–36).

By a broad tendon it inserts into the crest of the greater tuberosity and proceeds down the anterior aspect of the humerus for several inches. It passes over the bicipital groove and its tendon and the insertion of the subscapularis muscle tendon. It is in the medial and lateral thoracic nerves.

Its primary function is to pull the arm down from an overhead elevated

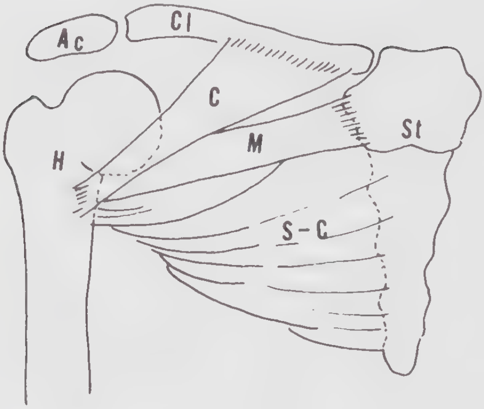


Figure 1–36. Pectoralis major. The pectoralis major has three laminae: clavicular (C), manubrial (M), and a sternocostal (S-C) bundle. All form the pectoralis muscle but are considered to have slightly separate functions. (Ac = acromion; Cl = clavicle; St = sternum.)

position as well as to adduct the arm and to rotate the humerus internally. It subsequently rotates the scapula upon the anterior rib cage in an anteromedial direction.

ACROMIOCLAVICULAR AND STERNOCLAVICULAR JOINT MOTION

In addition to gliding on the convex rib cage, the scapula circumducts about the outer end of the clavicle at the acromioclavicular joint (A-C).

The acromial aspect of the acromioclavicular joint is convex, articulating with the concave facet of the outer end of the acromium. A fibrocartilaginous ring resembling a meniscus exists in this joint at maturation. Postnatally this articulation is a synarthrosis. During development the constant rotational torque of the joint during arm motion causes tearing of the fibrous elements of the capsule joint structure, gradually forming the fibrous meniscus (Fig. 1-37).

No joint space essentially exists at age 2 years. The end of the clavicle and the acromium are connected by a fibrocartilaginous bridge. At approximately 3 years of age, joint spaces appear resembling two synovial cavities—one at the clavicular end and the other at the acromial tip. The remaining fibrous elements between these two cavities become a disk.

This disk becomes meniscoid by the second decade of life, and the articular surfaces of the acromium and the clavicle become smooth and glistening, resembling a synovial joint; it is essentially a pseudosynovial joint. After the second decade, rapid degenerative changes occur within this joint structure by virtue of the repeated rotatory traction forces expended by each upper arm motion.

The acromioclavicular joint has a weak thin relaxed capsule that is reinforced by strong superior and inferior acromioclavicular ligaments. These ligaments prevent posterior displacement of the clavicle upon the acromium.

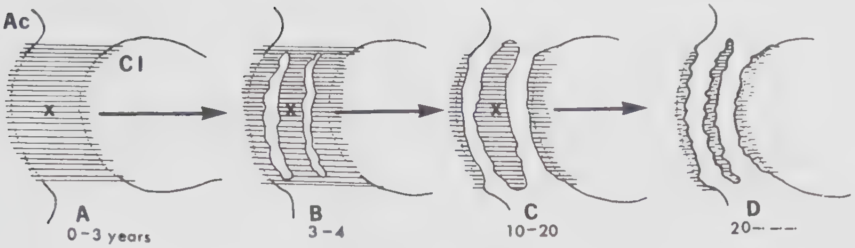

Ac
Cl
x
x
x
A
0–3 years
B
3–4
C
10–20
D
20— — —

Figure 1–37. Evolution of the acromioclavicular disk (meniscus). *(A)* From birth to age 2 a fibrocartilage bridge joins the acromion to the clavicle (Ac to Cl) with no joint space. *(B)* From age 3 to 4, cavities form on either side of what will become the meniscus (x). *(C)* In the first and second decades the meniscus is already beginning to thin and fibrillate, which increases rapidly from age 20 on. *(D)* In the sixth decade the meniscus may be completely gone.

The clavicle is firmly attached to the coracoid process of the scapula by the coracoclavicular ligaments (Fig. 1–38). These structures are resilient fascicles, each called a ligament—the laterally placed is termed the *trapezoid ligament* and the medially placed one is termed the *conoid ligament.* These ligaments attach the scapula firmly to the clavicle and prevent rotation in a coronal direction (Fig. 1–39).

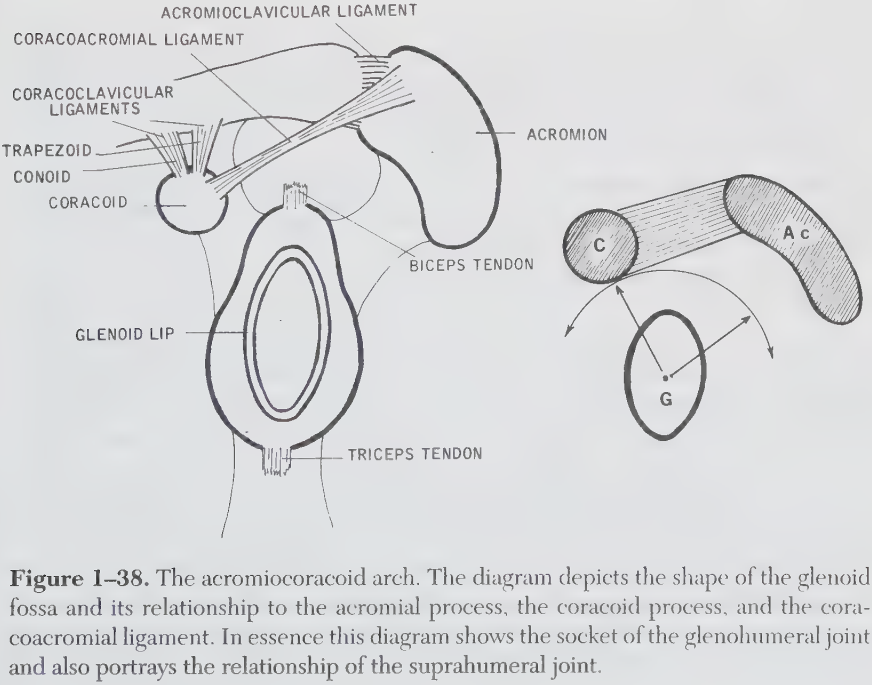

ACROMIOCLAVICULAR LIGAMENT
CORACOACROMIAL LIGAMENT
CORACOCLAVICULAR LIGAMENTS
TRAPEZOID
CONOID
CORACOID
ACROMION
BICEPS TENDON
GLENOID LIP
TRICEPS TENDON
C
Ac
G

Figure 1–38. The acromiocoracoid arch. The diagram depicts the shape of the glenoid fossa and its relationship to the acromial process, the coracoid process, and the coracoacromial ligament. In essence this diagram shows the socket of the glenohumeral joint and also portrays the relationship of the suprahumeral joint.

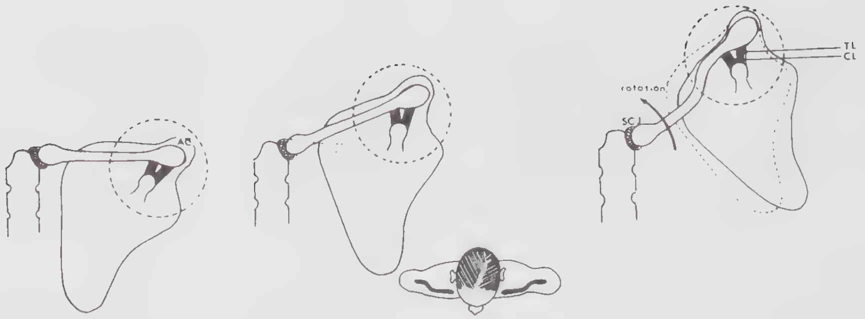


Figure 1–39. Action of the coracoclavicular ligaments upon the acromioclavicular joint. The coracoclavicular ligament attaches the scapula to the clavicle. It is divided into two resilient fascicles termed the *trapezoid ligament* and the *conoid ligament*. From the coracoid they proceed upward and laterally to attach onto the undersurface of the clavicle. Elevation of the clavicle without rotation maintains a constant relationship of the scapula to the clavicle. The rotation of the scapula depresses the coracoid and thus rotates the clavicle about its long axis. The left drawing depicts the scapula at rest with the coracoclavicular ligaments viewed through the sagittal axis *(dotted circle)*. The middle drawing shows abduction of the clavicle along the coronal plane without rotation. The right drawing shows full elevation of the clavicle, still displaying an unchanged relationship of the scapula to the clavicle in this coronal plane. Motion through this range must occur at the sternoclavicular joint (SCJ).

The trapezoid and conoid ligaments can, theoretically, support the scapula at the end of the strut furnished by the clavicle (Fig. 1–40). This has been refuted by the retention of stability of the clavicle after severance of these ligaments (Urist). Instability occurs only if there is severance of the coracoclavicular ligaments *and* the superior acromioclavicular ligament (SAC in Fig. 1–41). This support exists even though mechanical engineering principles would deny the strength of this latter ligament with its short lever arm.

Because the scapula must rotate externally about the A-C joint in the movement of overhead arm extension and through all its phases of forward flexion and abduction, the mechanical restraint of the claviculoscapular ligaments (see Fig. 1–39) is obviated by the crank shape of the clavicle and the fact that it rotates about its sternoclavicular joint. This clavicular shape and clavicular rotation permit the distal end of the clavicle to move at a great angle with a reasonably small proximal angulation (Fig. 1–41).

It has been calibrated that as the arm abducts past 90° in overhead elevation, the first 30° of clavicular elevation occurs at the sternoclavicular joint and the next 30° occurs as a result of the rotation of the outer aspect of the clavicle about its long axis.

The *sternoclavicular joint* is formed by the inner edge of the clavicle articulating about a fossa existing on the superior lateral margin of the *manubrium*

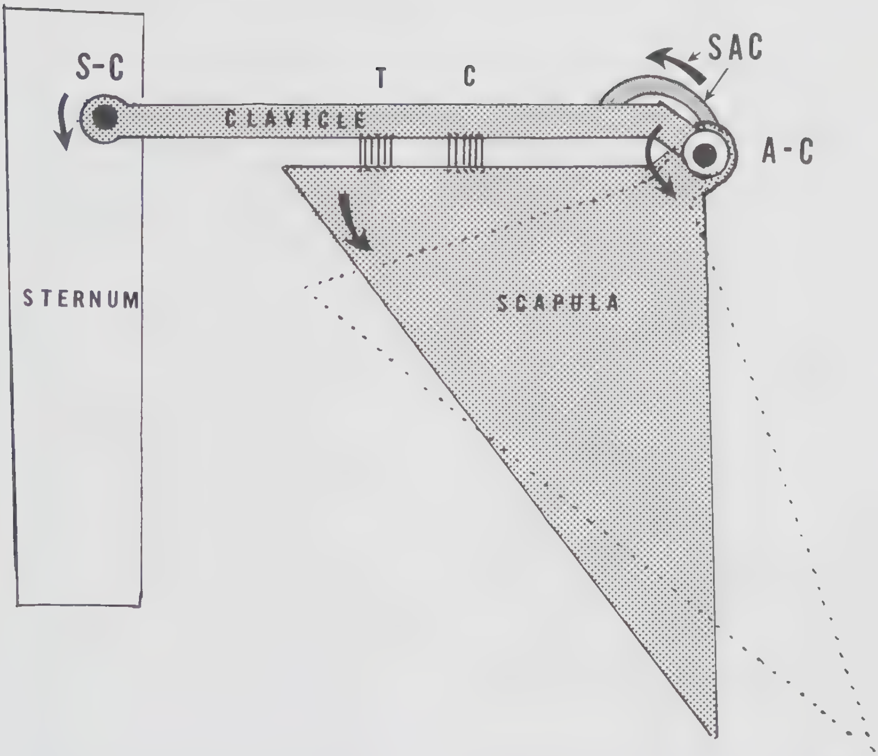


Figure 1–40. Static support of the scapula by claviculoscapular ligaments. The clavicle acts as a strut from the sternum at the sternoclavicular (S-C) joint. The scapula articulates upon the end of the clavicle at the acromioclavicular (A-C) joint. By its eccentric weight the scapula should rotate about the A-C joint, except for the restraint by the claviculoscapular trapezium (T) and conoid (C) ligaments. The superior acromioclavicular ligament (SAC) alledgedly supports the scapula upon severance of the T and C ligaments *(schematic drawing).*

sternum and the cartilage of the first rib (Fig. 1–42).

An articular disk exists between the fossa of the manubrium sternum and the clavicle which is attached to the medial aspect of the first rib. This disk (insert in Fig. 1–44) creates two joint spaces. This sternoclavicular (truly sternoclaviculocostal) articulation is stabilized by anterior and posterior sternoclavicular ligaments which connect the two (left and right) clavicles. There is also support from a strong ligament that arises from the medial portion of the first rib, which runs laterally and obliquely to attach to the undersurface of the extreme medial aspect of the clavicle (see Fig. 1–42).

The muscles acting upon the clavicle (Fig. 1–43) can be summarized as being the deltoid, trapezius, sternocleidomastoid, pectoralis major, subscapularis,

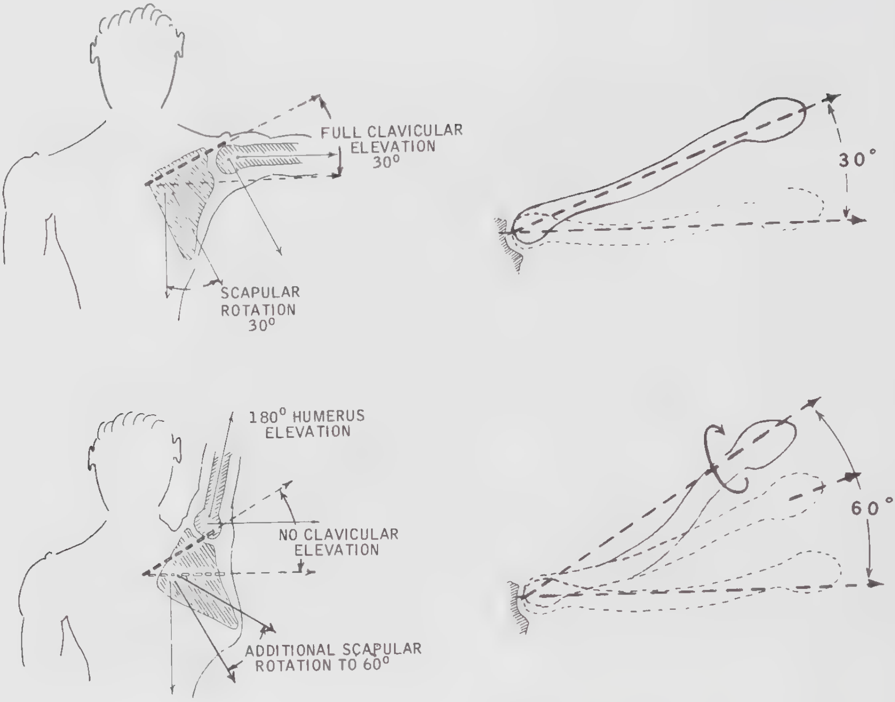

FULL CLAVICULAR ELEVATION 30°
SCAPULAR ROTATION 30°
30°
180° HUMERUS ELEVATION
NO CLAVICULAR ELEVATION
ADDITIONAL SCAPULAR ROTATION TO 60°
60°

Figure 1–41. Scapular elevation resulting from clavicular rotation. The upper drawing shows the elevation of the clavicle without rotation to 30°. The remaining 30° of scapular rotation, which is imperative in full scapulohumeral range, occurs by rotation of the crank-shaped clavicle about its long axis.

and, indirectly, all the muscles that attach to the scapula and the humerus.

Mechanically the sternoclavicular joint moves in a circumducting manner in all motions of the upper extremity, but, in spite of these frequent numerous motions, there occur little degenerative changes, late and minimal, as compared with the acromioclavicular joint.

SCAPULOHUMERAL RHYTHM MOVEMENT

To accomplish any arm movement, to place the hands and fingers in a functional position, both the scapula *and* the humerus must participate in a smooth, coordinated, essentially effortless, painless, and synchronous manner. Each joint must have adequate range of motion, implying normal articular cartilaginous surfaces. *All* involved muscles must be adequately innervated (extrafusal alpha innervation) from functional anterior horn cells in the cord. All spindle and Golgi systems must be operational and coordinated.

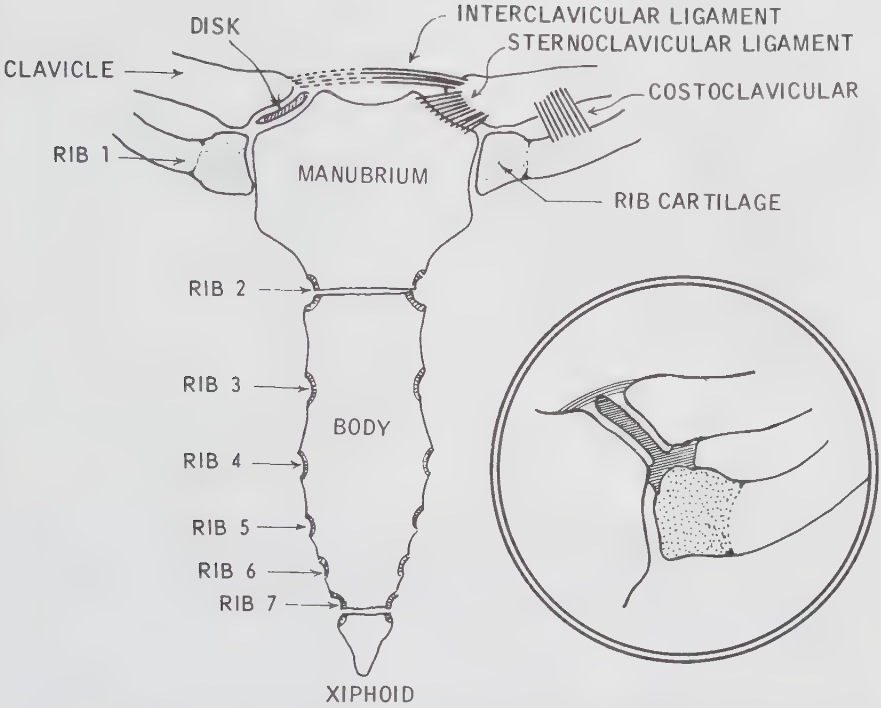

DISK
INTERCLAVICULAR LIGAMENT
STERNOCLAVICULAR LIGAMENT
CLAVICLE
COSTOCLAVICULAR
RIB 1
MANUBRIUM
RIB CARTILAGE
RIB 2
RIB 3
BODY
RIB 4
RIB 5
RIB 6
RIB 7
XIPHOID

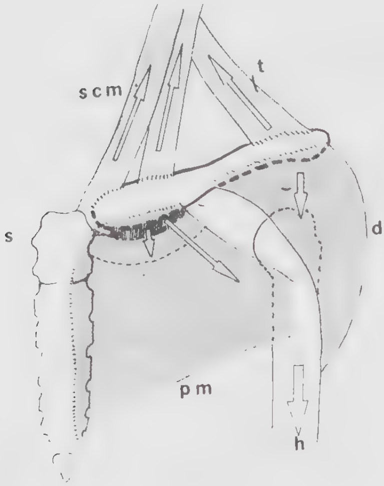
Figure 1–42. The sternoclavicular joint. The sternoclavicular joint is formed by the medial portion of the clavicle articulating upon the manubrium sterni and also with the cartilage end of the first rib. The ligaments that stabilize the joint are shown. The fibroelastic disk, or meniscus, is shown in the insert. In spite of marked movement at this joint in all shoulder girdle movements, arthritic changes are rare, mild, and rarely disabling.

In any clinical evaluation of abnormal painful movement of the shoulder girdle, the exact motion and *all its components* must be understood and its abnormal deviation from normal [must be] ascertained (Cailliet).

This integrated movement of the scapula and the humerus is termed *scapulohumeral rhythm* (Codman).

It has been simplistically stated that there is a 2:1 ratio of movement of the humerus to movement of the scapula in the process of abducting the arm. For every 15° of abduction of the arm, 10° occurs at the glenohumeral joint and 5° from rotation of the scapula upon the chest wall. This ratio allegedly occurs throughout the entire abduction range in a smooth coordinated pattern so that when the arm has reached 90° of abduction, 60° have occurred at the glenohumeral joint, and the scapula has rotated 30° (Fig. 1–44).

This rule has been refuted because the 2:1 ratio may not pertain at every degree of abduction. During the first 15° to 30° of abduction, for instance, the



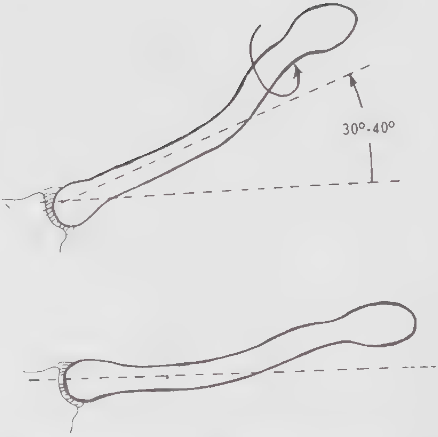


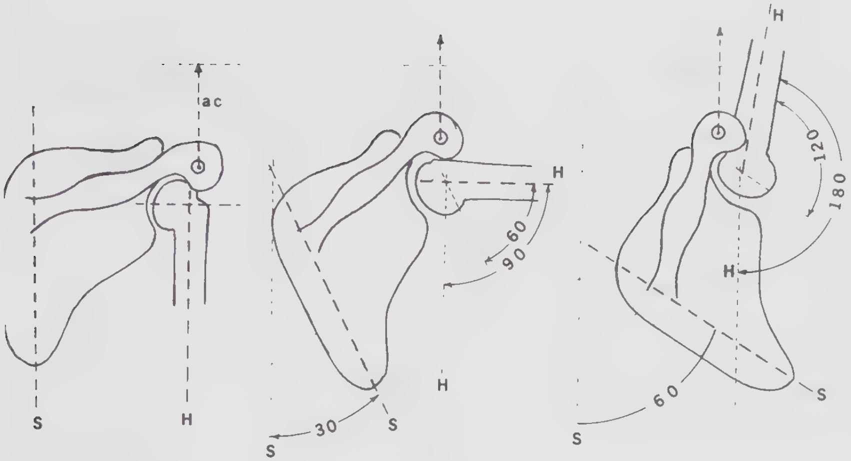
Figure 1–43. Muscles acting upon the clavicle. The major muscles acting upon the clavicle are shown, their direction of pull indicated by arrows: scm = sternocleidomastoid; t = trapezius; d = deltoid; s = subscapularis; and pm = pectoralis major. The gravity pull (h) of the arm itself is indicated as well. The muscles that act indirectly upon the clavicle are not shown.

scapula may remain *fixed* as a supporting base and move only at subsequent degrees of abduction. The most important aspect of the rhythm is that there is proportionate movement of both components and in an approximate 2:1 relationship. It is the coordinated movement, coupled with proportional rotation of the humerus, that results in physiologic arm motion.

The scapula rotates to alter the position of the overhanging acromium and coracoacromial ligament away from the rotating humeral head and its greater tuberosity and maintains the optimum lengthening of the deltoid muscle in the process of abduction (Fig. 1–45).

The deltoid muscle, like all muscles, has its greatest efficiency at its *rest length,* which is usually a point between maximum shortening and maximum elongation. In the deltoid muscle this midpoint is dependent at the side with the arm. The deltoid shortens (contracts) during abduction and reaches full shortening when the humerus has reached full abduction (90° with the arm in sagittal position, 60° with humerus internally rotated, and 120° with arm externally rotated).

As the arm abducts by simultaneous rotation of the scapula, the points of origin of the deltoid move in an arc, always maintaining some elongation of the deltoid. When the arm has reached full overhead extension, there is no need for deltoid contractions to support the arm, thus at this full shortened position efficiency of deltoid contraction is minimal.



$$\frac{S}{H} = \frac{30}{60} = \frac{60}{120} = \frac{1}{2}$$

Figure 1–44. Scapulohumeral rhythm. *(Left)* The scapula and the humerus at position of rest with the scapula relaxed and the arm dependent, both at position 0°. The abduction movement of the arm is accomplished in a smooth, coordinated movement during which for each 15° of arm abduction 10° of motion occurs at the glenohumeral joint and 5° occurs due to scapular rotation upon the thorax. *(Center)* The humerus (H) has abducted 90° in relation to the erect body, but this has been accomplished by a 30° rotation of the scapula and a 60° rotation of the humerus at the glenohumeral joint, a ratio of 2:1. *(Right)* Full elevation of the arm: 60° at the scapula and 120° at the glenohumeral joint.

With the arm fully extended overhead the glenoid fossa plane has reached almost horizontal position, which supports the head of the humerus. Only the capsule now maintains the head of the humerus within the glenoid fossa, which explains why this is the position of possible shoulder dislocation through the thin inferior aspect of the capsule and with no protection from the muscles of the shoulder complex.

As the scapula goes from a fixed stabilizing position, with the arm dependent at the side 0° position, to gradual rotation upon the thoracic wall, there is simultaneous proportionate motion of the glenohumeral joint (Fig. 1–46). This motion is abduction with slight downward gliding of the humeral head upon the glenoid fossa. Abduction of the humerus is accompanied by external rotation of the humerus about its longitudinal axis.

Whereas the rotator cuff muscles—the supraspinatus, infraspinatus, and teres minor—abduct the humerus, they also rotate the humerus externally, and the subscapularis muscle (an internal rotator) relaxes.

The scapular abductor rotator muscles—the trapezius and the serratus anterior—contract, and the opposing rhomboid and levator scapulae relax.

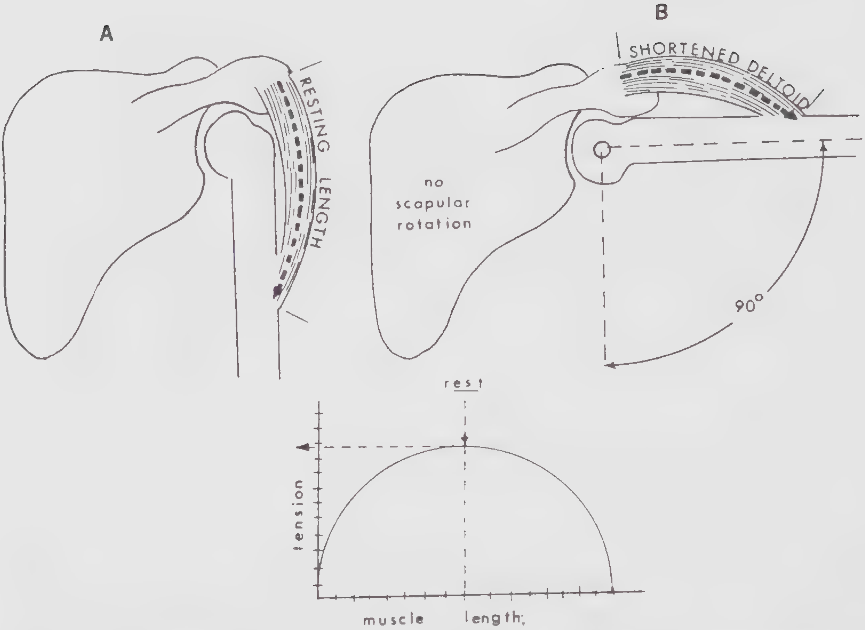


Figure 1–45. Deltoid action upon the glenohumeral joint. The mechanical efficiency afforded the deltoid action upon abduction of the humerus by the simultaneous rotation of the scapula is shown in the length-tension relationship with muscle. Muscle efficiency is greatest at rest length and diminishes upon shortening. In the abducted arm without scapular rotation, the deltoid shortens to a length of less tension (B). Simultaneous scapular rotation keeps the deltoid at rest length (A).

The pectoralis major muscle, also an internal rotator, relaxes as external rotation occurs.

The smooth, efficient, neuromuscular scapulohumeral action is a classic example of feedback from spindle-oriented controlled neurologic activity with intrinsic reciprocal relaxation. This coordinated activity must be *programmed* for every daily activity and for each athletic activity.

Every ligamentous articular aspect of the scapulohumeral joint complex must function approximately. The posture of the individual must also be considered in regard to arm function because the scapulothoracic joint is dependent on the vertebral posture. It is evident that the scapulohumeral rhythm is a very complex neuromuscular-articular functional activity.

THE BICEPS MECHANISM

The biceps is anatomically and functionally involved in the scapulohumeral mechanism of upper arm function. Its functional relationship to glenohumeral

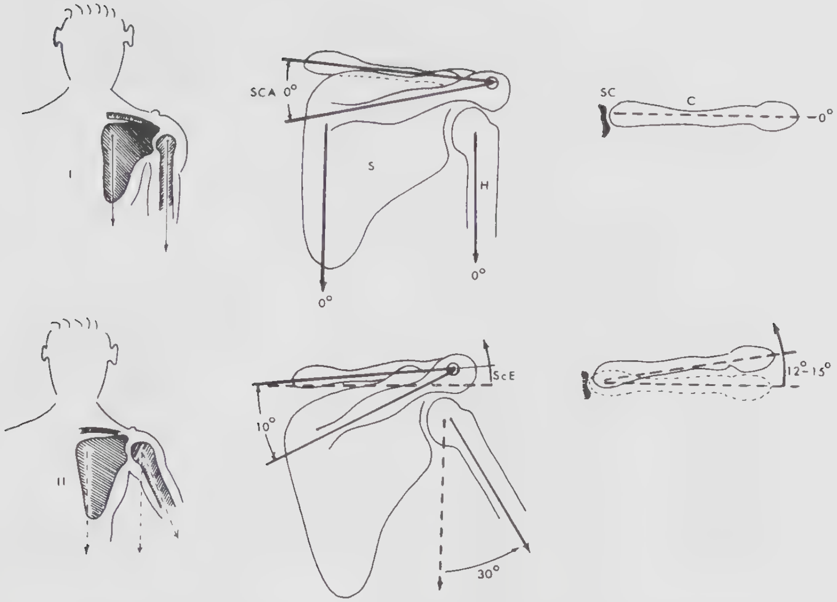


Figure 1–46. Accessory movement of the scapulohumeral rhythm other than the glenohumeral movement. Movement of the arm through all phases of abduction involves all joints of the shoulder girdle in a synchronous manner.

Phase I: The resting arm: 0° scapular rotation (S); 0° spinoclavicular angle (SCA); 0° movement at the sternoclavicular joint (SC); no elevation of the outer end of the clavicle (C); no abduction of the humerus (H).

Phase II: Humerus abducted 30°: the outer end of the clavicle is elevated 12° to 15° with no rotation of the clavicle; elevation occurs at the sternoclavicular joint; some movement occurs at the acromioclavicular joint as seen by an increase of 10° of the spinoclavicular angle (SCA) formed by the clavicle and the scapular spine.

motion is, interestingly, passive rather than active in regard to neuromuscular action.

The biceps brachii has two heads but a common tendinous insertion on the inner aspect of the radius. The short medial head originates from the coracoid process. The long head originates from the superior lip of the glenoid fossa. Its tendon of origin, the long head, proceeds laterally and angles 90° at the bicipital groove of the superior humeral head. It then proceeds downward to merge in the common muscle belly.

Because of the attachment of the biceps on the ulnar side of the radius, the primary function of that muscle is supination of the forearm, and the secondary function is elbow flexion. By its origin and insertion in the anterior aspect of the upper arm, it assists the anterior deltoid fibers in forward flexion of the shoulder.

There is no motion of the biceps tendon within the bicipital groove when

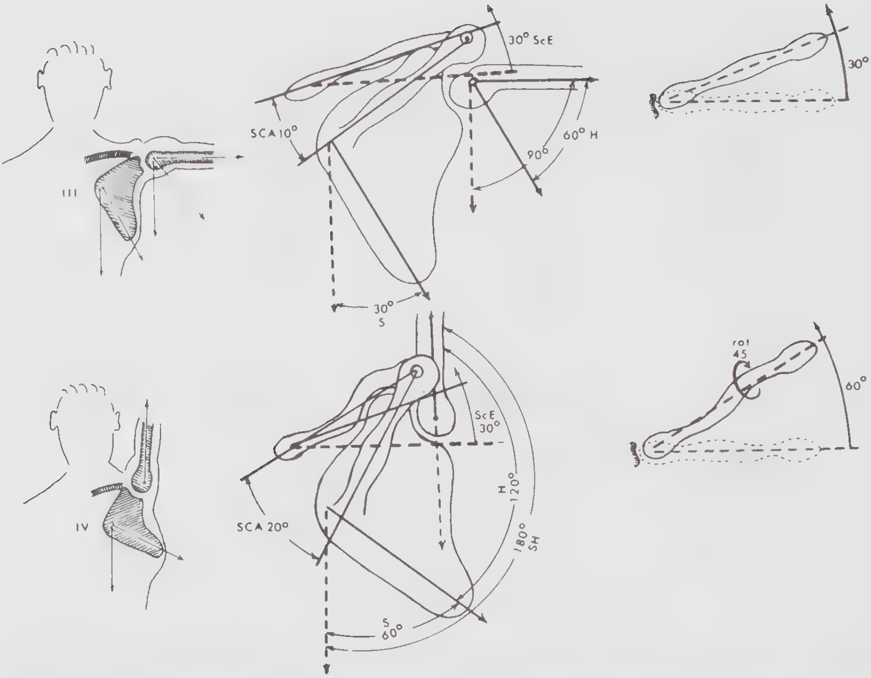


Figure 1–46 *(continued).* *Phase III:* Humerus (H) abducted to 90° (60° glenohumeral, 30° scapular); the clavicle is elevated to its final position, 30°; no rotation of clavicle has occurred (all movement is at the sternoclavicular joint); no change in the SCA.

Phase IV: Full overhead elevation (SH = 180°; H = 120°; S = 60°): outer end of the clavicle has not elevated further (at the sternoclavicular joint), but the SCA has increased (to 20°). Because of the clavicle's rotation and its cranklike form, the clavicle elevates an additional 30°. The humerus through this phase has rotated, but this has not influenced the above degrees of movement.

the biceps contracts to supinate the forearm or to flex the elbow with the humerus in the dependent position. At best, contraction of the biceps muscle presses down into the bicipital groove and depresses the entire humerus.

There is motion of the tendon within the bicipital groove only when the bicipital groove moves on the tendon during humeral motion. The tendon glides within the groove and is held there by the transverse humeral ligament. As the arm rotates internally, the tendon rolls within the groove and moves downward. As the arm abducts higher than horizontal, the tendon glides within the bicipital groove (Fig. 1–47) and is held within the groove by the transverse humeral ligament.

By its passive action upon the humerus, abduction of the humerus upon the glenoid fossa, which occurs, with downward glide simultaneously with external rotation, occurs passively with contraction of the long head of the biceps.

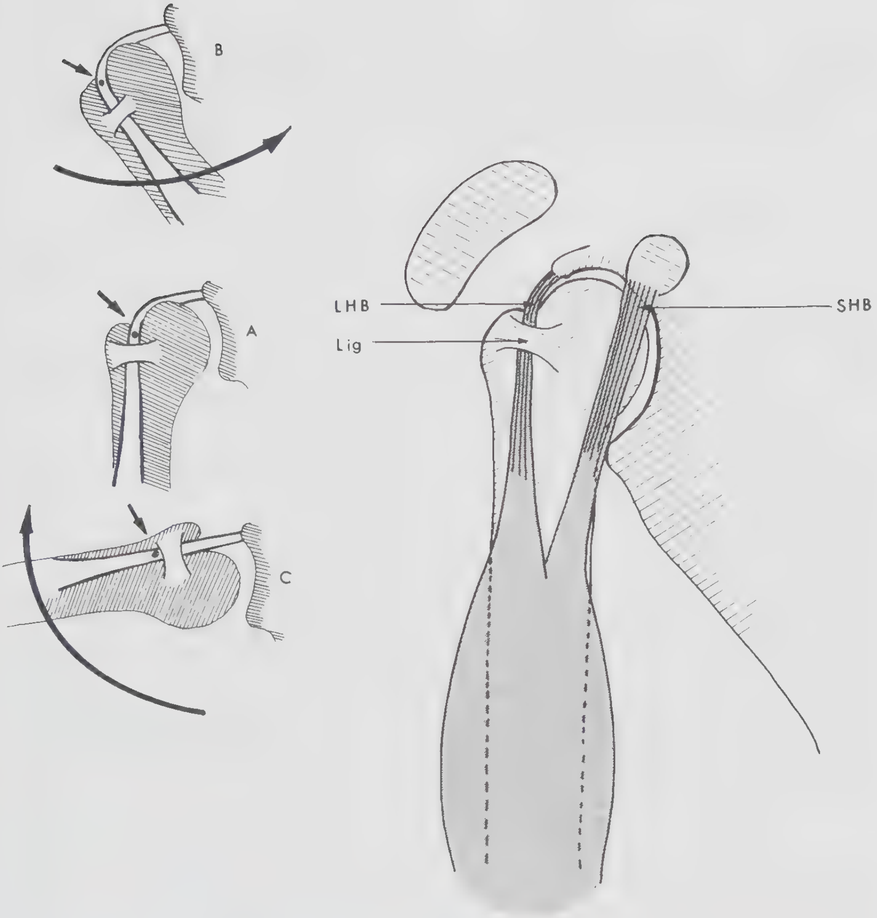


Figure 1–47. Biceps mechanism. The biceps brachii originates from two tendons: the short medial head, from the coracoid process; and the long head, from the superior rim of the glenoid fossa. The long head passes down into the bicipital groove in a fibrous sheath between the tendons of the subscapularis and the supraspinatus tendon. The small drawings at left depict the movement of the humerus upon the biceps tendon. *(A)* The dependent hanging arm. *(B)* Arm adducted, internally rotated, and extended, causing the ligament *(dot)* to move away from the transverse humeral ligament. *(C)* The downward movement of the ligament *(dot)* below the transverse humeral ligament when the arm is abducted, externally rotated, and flexed forward.

The downward pull on the greater tuberosity of the infraspinatus and the teres minor muscles also depresses the humerus, but the greatest downward glide has been attributed to the mechanical force of the tense bicipital tendon.

The relationship of the biceps tendon, within the bicipital groove, to the attachment of the coracohumeral ligament, the supraspinatus tendon, the subdeltoid bursa, and the upper anterior aspect of the glenohumeral capsule indicates why, clinically, a *tendinitis* of the conjoined tendon complicates the entity of bicipital tendinitis in the painful shoulder.

BIBLIOGRAPHY

Cailliet, R: Mechanics of Joints. In Arthritis and Physical Medicine, (ed. Licht) Elizabeth Licht Publishers, New Haven, 1969.

Codman, EA: The Shoulder. Thomas Todd, Boston, 1934.

DePalma, AF: Surgery of the Shoulder, ed 2. JB Lippincott, Philadelphia, 1973.

Inman, VE, Saunders, JB, and Abbott, LC: Observations of the function of the shoulder joint. J Bone Joint Surg 26:1, 1944.

MacConnaill, MA: Studies in the mechanics of synovial joints. Irish J Med Sci 21:223, 1946.

MacConnaill, MA: The movement of bones and joints. J Bone Joint Surg 32B:244, 1950; 33B:251, 1951.

McGregor, L: Rotation at the shoulder: Critical inquiry. Br J Surg 24:425, 1937.

Mennell, J: The Science and Art of Joint Manipulation, Vol 1. J & A Churchill, London, 1949.

Moseley, HF: Shoulder Lesions, ed 3. Churchill Livingstone, Edinburgh, 1969.

Saario, L: The range of movement of the shoulder joint at various ages. Acta Orthop Scand 33(4):366, 1963.

Simon, WH: Soft tissue disorders of the shoulder. Orthop Clin North Am 6(2):521, 1975.

Thomas, CL (ed): Taber's Cyclopedic Medical Dictionary, ed 16. FA Davis, Philadelphia, 1989.

Urist, MR: Complete dislocations of acromioclavicular joint. J Bone Joint Surg 28:813, Oct., 1946.

Van Linge, B and Mulder, JD: Function of the supraspinatus muscle and its relation to the supraspinatus syndrome. J Bone Joint Surg 45(B):750, 1963.

CHAPTER 2

Tissue Sites and Mechanisms of Shoulder Girdle Pain

The tissue sites and causes of pain in the shoulder complex are numerous, as depicted in this outline:

I. **Musculoskeletal**
   A. Degenerative
      1. Tendinitis, with or without calcific deposits
      2. Cuff tear, partial or complete
   B. Traumatic
      1. Fracture
      2. Dislocation
      3. Acromioclavicular separation
      4. Biceps tendon tear
   C. Inflammatory
      1. Rheumatoid arthritis
      2. Gout
      3. Infectious arthritis
   D. Tumors
      1. Bone
      2. Soft tissue

II. **Neurologic**
   A. Peripheral nerve
      1. Root (cervical)
         a. Spinal foraminal
            (1) Spondylosis

            - (2) Herniated disk
            - (3) Traumatic
                - (a) Fracture
                - (b) Dislocation
        - b. Extramedullary tumors
    - 2. Brachial plexus
        - a. Mechanical
            - (1) Neurovascular bundle compression (*cervical dorsal outlet syndromes*)
            - (2) Scalene anticus syndrome
            - (3) Cervical rib
            - (4) Claviculocostal syndrome
            - (5) Pectoralis minor syndrome
        - b. Trauma
            - (1) Traction or penetrating injuries
        - c. Inflammatory
            - (1) Brachial plexitis
        - d. Tumors
            - (1) Pancoast
            - (2) Adenitis
- B. Central nervous system
    - 1. Intramedullary tumors
    - 2. Syringomyelia

III. Vascular

- A. Arterial
    - 1. Occlusive: acute and chronic
        - a. Embolic
        - b. Vasospastic
        - c. Traumatic
        - d. Atherosclerotic
    - 2. Aneurysm or fistula
- B. Venous
    - 1. Phlebitis
- C. Lymph
    - 1. Lymphedema

IV. Referred Visceral-Somatic Pain

- A. Cardiac
    - 1. Anginal pain
    - 2. Hand-shoulder syndrome
        - a. Causalgia
- B. Gallbladder
- C. Diaphragmatic

D. Ruptured viscus

V. **Articular**
   A. Degenerative
   B. Inflammatory
   C. Infectious
   D. Metabolic

Numerous tissues are potential sites of nociception. When injured, these tissues cause functional impairment as well as pain. The prime purpose of the shoulder girdle is to allow for placement of the hand in a functional position. It can become impaired in its function either by physical damage or from pain.

Every one of the seven or more joint structures within the shoulder complex is capable of being a site of nociception or functional impairment. (Fig. 2–1). These joints can be enumerated as well as can the tissues within their confines as sites of pain and malfunction:

1. Glenohumeral joint
2. Suprahumeral joint
3. Acromioclavicular joint
4. Sternoclavicular joint
5. Scapulothoracic joint
6. Costosternal joint
7. Costovertebral joint
8. Bicipital tendon
9. Rotator cuff conjoined tendon

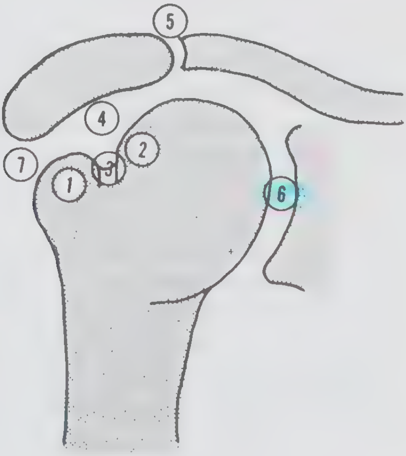


Figure 2–1. Sites of tissue pain: (1) greater tuberosity: attachment of supraspinatus tendon; (2) lesser tuberosity; (3) bicipital groove: tendon of long head of biceps; (4) subacromial bursa; (5) acromioclavicular joint; (6) glenohumeral joint and capsule; (7) subdeltoid bursa. (Modified from Steindler, A: The Interpretation of Pain in Orthopedic Practice. Charles C. Thomas, Springfield, Illinois, 1959.)

10. Subdeltoid bursa
11. Glenohumeral capsule

Any one or all of these tissue sites are potentially the major site of nociception and impairment, but the major area of pathologic process is (1) the glenohumeral joint (Fig. 2–2) of the shoulder complex and (2) the supraspinatus tendon in that region with the contiguous bursal tissues of the subdeltoid region and the supralateral aspect of the glenohumeral capsule. Admittedly, all the other sites noted above have potential for pain and impairment, but the daily use, abuse, and misuse of the glenohumeral joint makes it the most vulnerable. The tissue within the glenohumeral joint most prone to pain and impaired function is the supraspinatus tendon.

TENDINITIS: TRAUMA, ATTRITION, AND DEGENERATION

The human being's upright posture and marked mobility of the shoulder girdle complex in activities of daily living are conducive to degeneration of the supraspinatus tendons and all the contiguous tissues. The joint structures and musculature are so created and structured as to give great mobility and adequate stability. The erect two-legged stance eliminates the excessive weight bearing upon the shoulder girdle, but the need to place the hands in so many regions and positions demands excessive range of motion of the shoulder, arm, forearm, and wrist. Stability of the shoulder girdle has been sacrificed for this mobility.

In a passive stage with the arm totally dependent, the effect of gravity imposes its stress upon the supraspinatus tendon. The supraspinatus muscle maintains its adequate tone through spindle activity, which must be constant. Fatigue may well play a role here in ultimate degeneration, impairment, or pain.

Any weight carried by the hands in this vertical dependent position of the arm initiates appropriate commensurate muscular contraction of the supraspinatus and the deltoid muscles. Again, muscular fatigue may result.

The mechanical effect of muscular contraction is mediated through the tendon of that muscle in its attachment to the corresponding bones. The supraspinatus tendon is angled toward its attachment to the greater tuberosity rather than directly to the bone. Traction on the tendon causes a relative ischemia by mechanical compression of the intrinsic blood vessels during contraction. This tension is compounded by the direct pull of the muscle on the tendon and the tethering impounded by its angulation. The area that becomes compressed within the supraspinatus tendon is termed the *critical zone*.

Theoretically, prolonged tension on the tendon by prolonged carrying of weights with the dependent arm could cause ischemic degeneration. This has not been confirmed as have other factors of ischemia in daily activities, but sus-

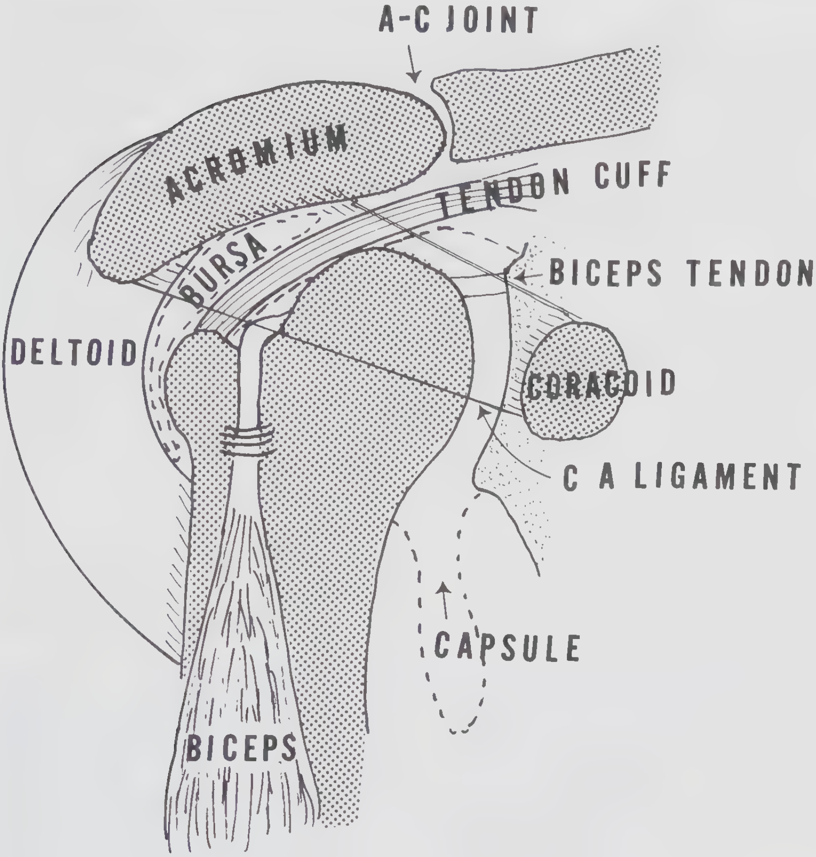


Figure 2–2. Tissue within the suprahumeral joints space. Between the overhanging acromium and the acromioclavicular joint are the supraspinatus tendon and its sheath. Under the deltoid muscle is the subdeltoid bursa. This bursa has a synovial lining that covers the undersurface of the acromium and the undersurface of the deltoid muscle. It pleats around to cover the greater tuberosity and the supraspinatus tendon. The glenohumeral joint capsule coats the undersurface of the rotator cuff, the glenoid labrum, and the inner surface of the upper humerus. It is invaginated by the tendon of the long head of the biceps. All these tissues are contained within a relatively shallow space also covered by the coracoacromial ligament.

tained isometric contraction of the supraspinatus muscle has been implicated as a cause of muscular degeneration (Hagberg, 1981). This stress on the supraspinatus muscle has been claimed as prevalent in postural occupational diseases. When a muscle loses its integrity, its tendon also is exposed to variation in the sustained tonus, and pathologic changes may occur.

Shoulder tendinitis is frequent and has been considered to be present in

18 percent of shipyard welders in one Swedish study (Heberts and colleagues). The predisposing factor leading to tendinitis is degeneration caused by nutritional deprivation (ischemia) and mechanical stress. Within the tendon there occurs formation of debris-containing calcium and breakdown of fibrils.

Microruptures of the fibrils result in debris and inflammation. Early microscopic changes (McLaughlin) reveal changes in the hyalin of the collagen fiber. Gradually the tendon fibrils (collagen) become fibrillated. As further degeneration occurs, the tendon strands break free and get ground down, resembling necrotic debris. Some calcium is deposited as microscopic crystals, causing inflammatory reaction with the presence of giant cells. This breakdown occurs mostly or predominantly in the area of avascularity, which has been postulated within the supraspinatus tendon (MacNab).

Circulation within a tendon is inversely proportional to the tension and actually decreases with increased tension (Schatzner and Banemark). Prolonged tension from and upon the dependent arm causes ischemia, and sustained isometric muscular contraction also sustains tension within the tendon.

Repetitive arm elevation at the glenohumeral joint, therefore, also causes repeated tension within the tendon. Laboratory studies upon humans and rabbits have revealed that inflammation of a tendon can result from repetitive contractions. This, in part, is also due to edema and deposition of fibrin in the paratenon as well as in the muscle interstitium (Rais)

Many daily and prolonged positions of the arm are with the arms held ahead of the body. This posture occurs in standing and sitting positions. The effort causes sustained isometric contraction of the supraspinatus and deltoid muscles as well as of the scapular muscles. Supraspinatus fatigue may occur, and because the supraspinatus has a longer tendon and the other muscles of the shoulder girdle have no significant tendon attachment to the bones involved, this muscle undergoes greater degenerative changes than do the other muscle tendons.

A progressive degeneration of the rotator cuff has been noted in people unaware of pain or discomfort (DePalma). It is well documented that any person past the age of 65 will demonstrate rotator cuff degeneration, fibrillation, and even partial if not complete tearing. All are, obviously, not necessarily symptomatic but pathologic changes; nevertheless, they are present.

Tearing, fibrillation, and degeneration occur mostly in the critical zone (see Fig. 1–25). Degenerative changes in tendons that resembled those of the rotator cuff were experimentally produced (MacNab). MacNab considered these degenerative changes to be secondary to hypovascularity, which he claimed was caused by mechanical compression.

There is also noted a wearing away of the tuberosities of the glenohumeral joints at the sites of tendonous attachment (Fig. 2–3), indications of the overall degenerative changes. These changes are incidentally noted in routine x-ray studies of the shoulder girdle of elderly people and do not necessarily implicate these sites as the causation of symptoms.

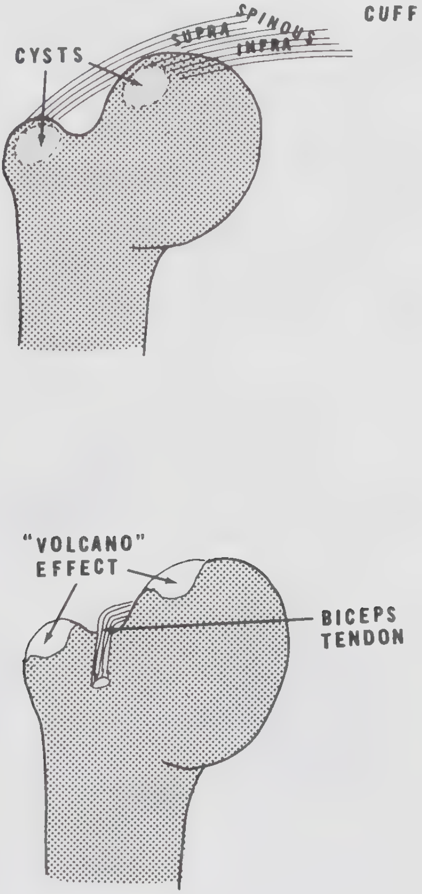


Figure 2–3. Roentgenographic changes in shoulder dysfunction: cysts in the tuberosities of the humerus. These are early x-ray evidence of attrition. With wear and tear, the tuberosities become eroded, causing a volcano appearance. The bicipital groove becomes shallow.

The wearing away of tuberosities about the bicipital groove also makes the fossa, which contains the long head of the biceps tendon, more shallow and thus gives less support to the tendon in its function.

Aging particularly affects the supraspinatus cuff tendon. The reason is based on the vascular supply and its constant exposure to stress manifested as traction, compression, or friction.

The tension of the dependent static arm has been described. The kinetic aspect of the arm during abduction and overhead elevation is probably more traumatic. When the arm is usually elevated, abducted, or held in the overhead position for many hours a day, there is compression stress during those hours as the greater tuberosity passes by, under, and behind the overhanging acromium and the coracoacromial ligament.

The rounded-shoulder posture of aging plays a further traumatic effect upon the rotator cuff. This will be discussed in Chapter 5. Rounded shoulder posture also develops during activities of daily living and in most desk jobs (Figs. 2–4, 2–5). Standing posture over a long period is also traumatic (Fig. 2–6).

Mechanical trauma is also chemical. There is a release of chemicals during physical trauma, and there are vascular sequelae (Fig. 2–7). In addition to prostaglandins, there are numerous other chemicals, including substance P, kinins, and histamine. New chemical mediators of pain are being identified frequently.

Tissue trauma also causes vascular changes that explain the original definition of inflammation: pain, swelling, redness, and tenderness. One sequence of trauma is depicted in Figure 2–8. Any of these traumatic consequences upon

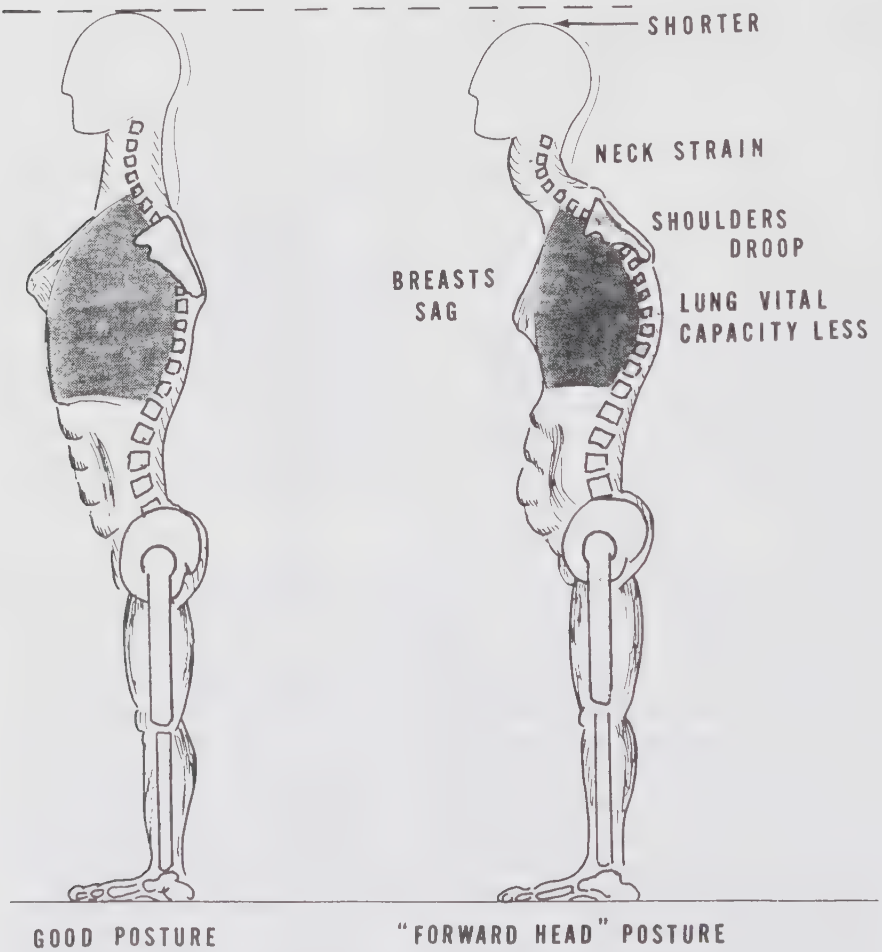

SHORTER
NECK STRAIN
SHOULDERS DROOP
BREASTS SAG
LUNG VITAL CAPACITY LESS
GOOD POSTURE
"FORWARD HEAD" POSTURE

Figure 2–4. Forward-head posture.

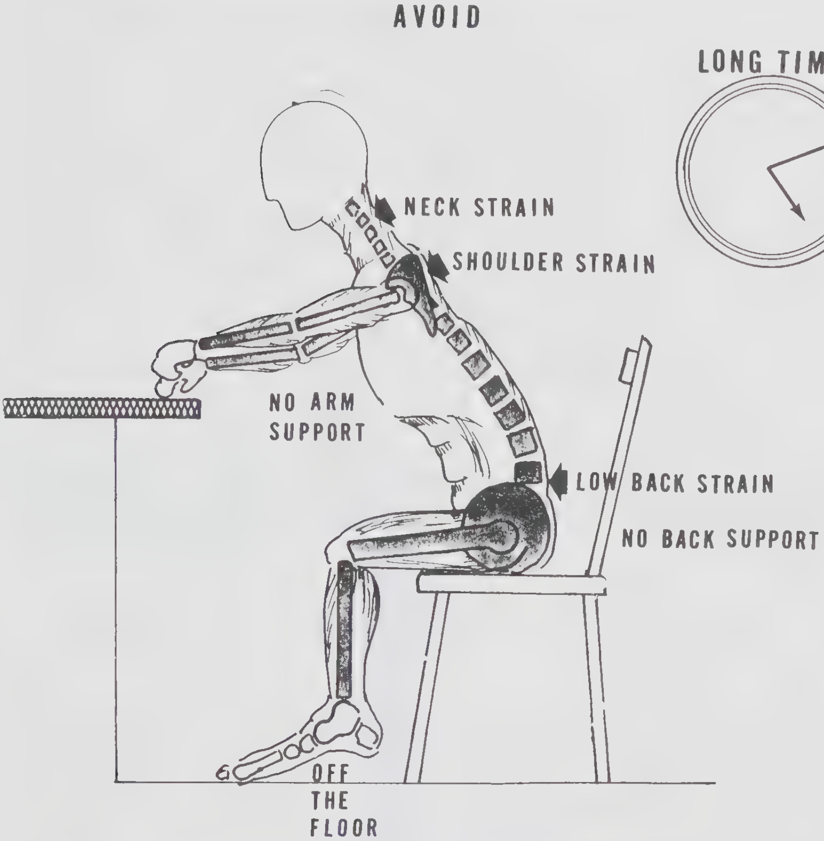

AVOID
LONG TIME
NECK STRAIN
SHOULDER STRAIN
NO ARM SUPPORT
LOW BACK STRAIN
NO BACK SUPPORT
OFF THE FLOOR

Figure 2–5. Faulty occupational posture: strain from sitting for a long period of time.

the supraspinatus tendon can result in (1) local pain, (2) local swelling, (3) entrapment and potential painful arc as the result of swelling in the confines of the suprahumeral space (4) compression of the tendon, especially of the critical zone, causing alternating hyperemia and hypemia with consequent fibrillation and necrosis. This sequence is depicted in Figure 2–9.

Neer described a continuum of stages as the *impingement syndrome*, listed below, in which he ascribed impingement as responsible in 95 percent of tears rather than circulatory impairment.

Stage 1: Edema and hemorrhage
Stage 2: Fibrosis and tendinitis
Stage 3: Bone spurs and tendon rupture

These arbitrary stages can be disputed, inasmuch as impingement is a form of

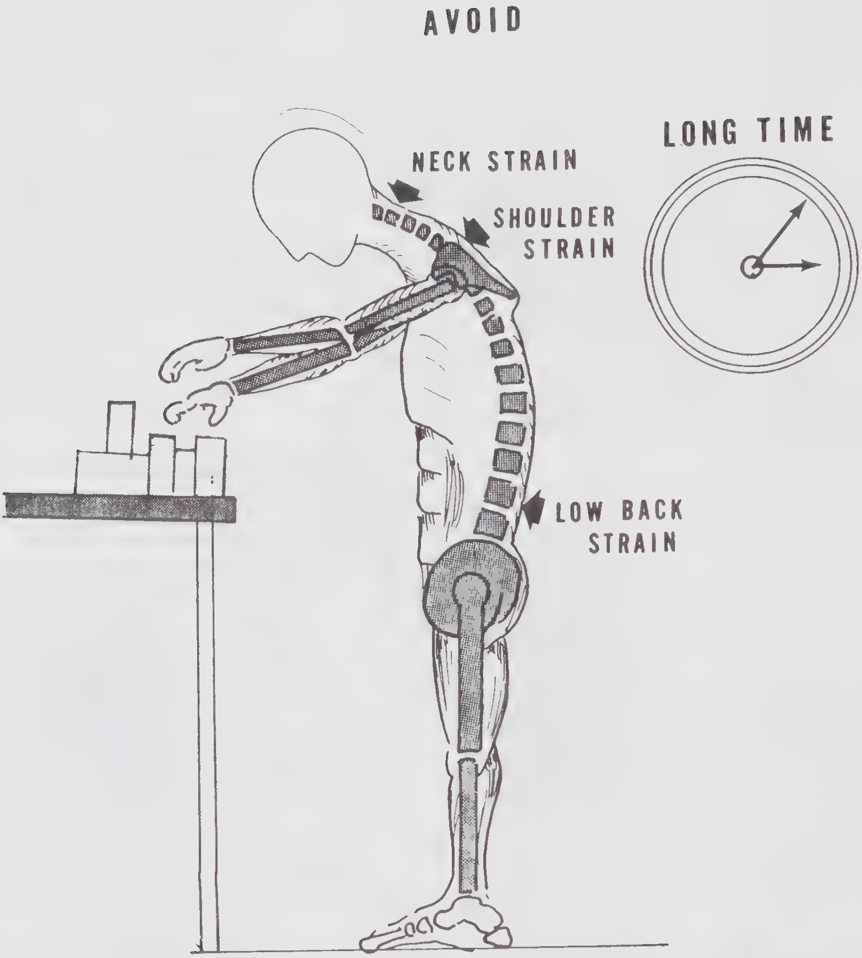

AVOID
LONG TIME
NECK STRAIN
SHOULDER STRAIN
LOW BACK STRAIN

Figure 2–6. Faulty occupational posture: strain from standing for a long period of time.

trauma that initiates or accompanies ischemia, and thus the two are significantly related.

SYMPTOMS OF SUPRASPINATUS TENDINITIS

Often the patient presents with a vague history of pain in the anterior shoulder region (see Fig. 2–1), but frequently there is no clear reason as to when, why, or how it occurred. A direct fall upon the outstretched arm may be

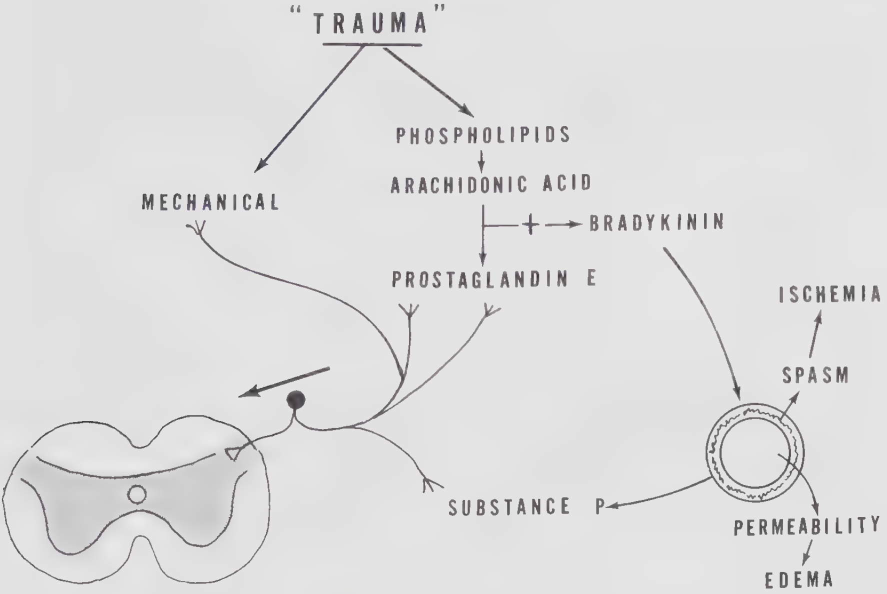

"TRAUMA"
PHOSPHOLIPIDS
ARACHIDONIC ACID
MECHANICAL
+
BRADYKININ
PROSTAGLANDIN E
ISCHEMIA
SPASM
SUBSTANCE P
PERMEABILITY
EDEMA

Figure 2–7. Chemical mediation of chemical nociceptors from trauma.

the initiating factor and will be recalled, as will a direct blow on the shoulder from the front or overhead.

Abrupt overhead arm elevation in a faulty manner may be elicited from the patient's history when the incident and its significance have not been considered meaningful. For example, raising a garage door—which requires abduction and forward flexion with simultaneous external rotation—may have been done

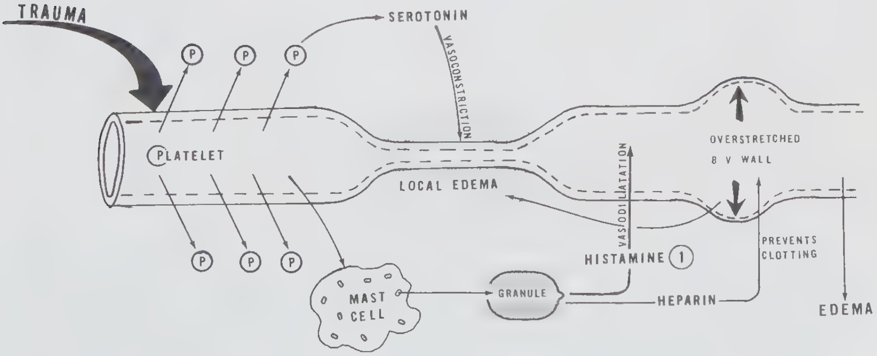

TRAUMA
SEROTONIN
P
P
P
VASOCONSTRICTION
PLATELET
OVERSTRETCHED
B V WALL
LOCAL EDEMA
VASODILATATION
P
P
P
HISTAMINE ①
PREVENTS CLOTTING
MAST CELL
GRANULE
HEPARIN
EDEMA

Figure 2–8. Vascular sequelae of tissue trauma.

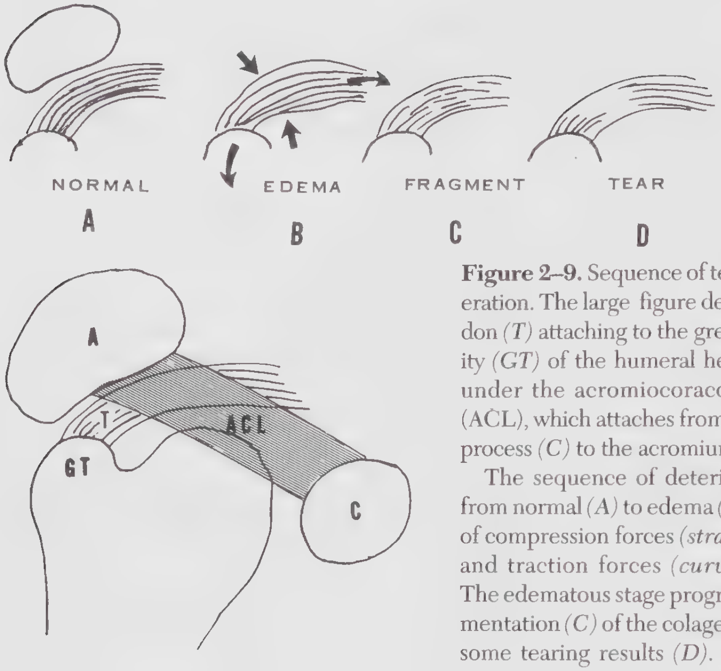


Figure 2–9. Sequence of tendon degeneration. The large figure depicts the tendon *(T)* attaching to the greater tuberosity *(GT)* of the humeral head. It passes under the acromiocoracoid ligament (ACL), which attaches from the coracoid process *(C)* to the acromium *(A)*.

The sequence of deterioration goes from normal *(A)* to edema *(B)* as a result of compression forces *(straight arrows)* and traction forces *(curved arrows)*. The edematous stage progresses to fragmentation *(C)* of the colagen fibers until some tearing results *(D)*. Tearing may be partial or total. The straight arrows show the compression occurring between the greater tuberosity and the acromium and the coracoacromial ligament. (See text for details.)

without adequate external rotation. This causes the greater tuberosity to impinge on the acromium. Lifting an object overhead without adequately extending the upper body may also be a factor.

Faulty body mechanics during athletic activities (the techniques of serving, pitching, or bowling) may cause injury. Fatigue from *overuse* may impair an otherwise well-programmed technique. A distraction may also momentarily impair an otherwise correct motion.

Regardless of the cause, the patient complains of pain in the shoulder area. Tenderness over that area may be claimed, demonstrated by the patient and confirmed by the examiner.

Passive range of motion may be painful, especially if it occurs during the painful arc. Active abduction of the upper arm may be limited throughout the entire range of motion if there is apprehension by the patient, protective spasm, or a moderately inflamed swollen tendon.

Pain and restricted motion may be reproduced only upon reaching the painful arc. This *painful arc* is the motion from approximately 60° of abduction to over 90°, going up or coming down. It is during this range that the greater tuberosity passes under the acromium and the coracoacromial ligament. If there is pain at this juncture, it indicates that the tendon is swollen, erythematous, and inflamed. There is insufficient room in the suprahumeral joint space for the tendon to proceed.

The painful arc is a term used, essentially, in abduction toward overhead elevation, but there is also a painful arc in external rotation at the time the greater tuberosity passes the acromium or the coracoacromial ligament.

In the case of tendon tearing, the painful arc may be noted clinically. In a complete tear, the painful arc is not experienced because there is *no* possible active abduction or external rotation. In the completely torn tendon there is also no significant bulging of the affected tendon. It is in the partially torn rotator tendon that the painful arc can be noted.

External rotation of the arm in the dependent position or with the arm abducted should be possible, albeit painful and limited, *if the cuff tendon is not completely severed.* Contracting the external rotators in the presence of an acutely inflamed tendon and partially torn tendon usually causes pain and discomfort, but some motion is usually possible.

If there is question as to whether there is a tear or even as to the extent of a tear, an injection of an anesthetic agent into the suprahumeral joint space will permit active and passive abduction and external rotation after the acute pain has been diminished. With the pain diminished, it becomes easier to determine the presence of a complete tear but not necessarily to make a differential diagnosis between tendinitis and a partial tear and its extent.

Modifying the posture also helps ascertain the degree of the painful arc (Fig. 2–10). It has been determined that posture alters the normal physiologic range of glenohumeral joint motion. Rounded shoulder posture decreases the range mechanically. It stands to reason, therefore, that change of posture will increase or decrease the subjective and extent of the painful arc. With the patient intentionally slumping forward, the arc will be greater and more painful, whereas with an attempted erect posture the arc may be decreased.

Tenderness can be elicited by pressure on areas 1, 2, 3, 7, and 4 in Figure 2–1, as well as on tissues within these areas. Tenderness over the A-C joint (5) would imply irritation to that joint.

CALCIFIC TENDINITIS

As mentioned earlier in the discussion on tendon degeneration, calcium crystals are often deposited within the nidus of collagen hyalin debris. When these particles are small, they are not visible on roentgenograms, but they have been claimed to be present in most people by age 35 (Decker). They are apparently kept in solution and thus are asymptomatic. This implies that there is some hyperemia of the critical zone as well as periods of relative ischemia.

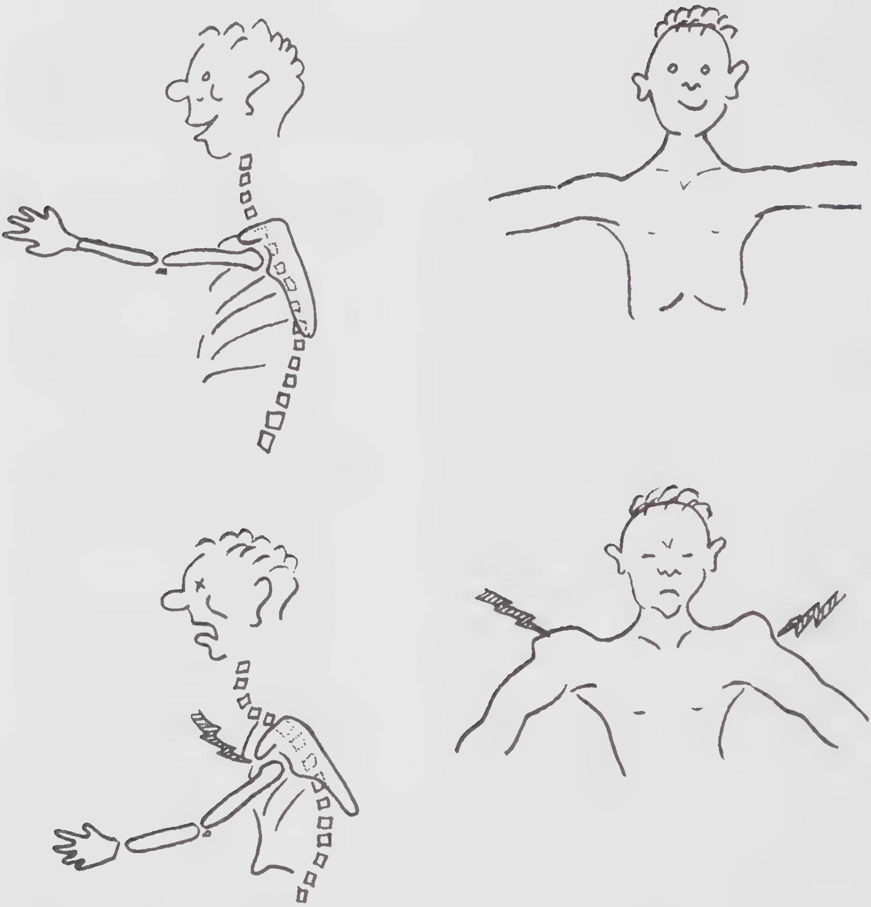


Figure 2–10. Postural effect upon glenohumeral range of motion. The upper drawings show the erect, *good* posture of the young: the forward elevation and the lateral abduction range of the arm is full and free; the coracoacromial ligament is elevated, and the humerus can do its corrective external rotation to allow the greater tuberosity to pass under the acromion. The lower drawings depict the dorsal kyphotic posture of the aged, the depressed individual, or the occupationally stressed person, in which the coracoacromial hood is lowered and the arm is internally rotated. Both of these factors cause impingement of the humerus against the arch. Compression and attrition of the cuff tendons result.

When there is build-up of calcium or a greater dehydration of these crystals, they become visible on roentgenograms. The hydrated calcium may also initiate pain and further impairment. Trauma superimposed on an existing calcific tendinitis may cause hyperemia that increases the content of the liquid calcium (Fig. 2–11).

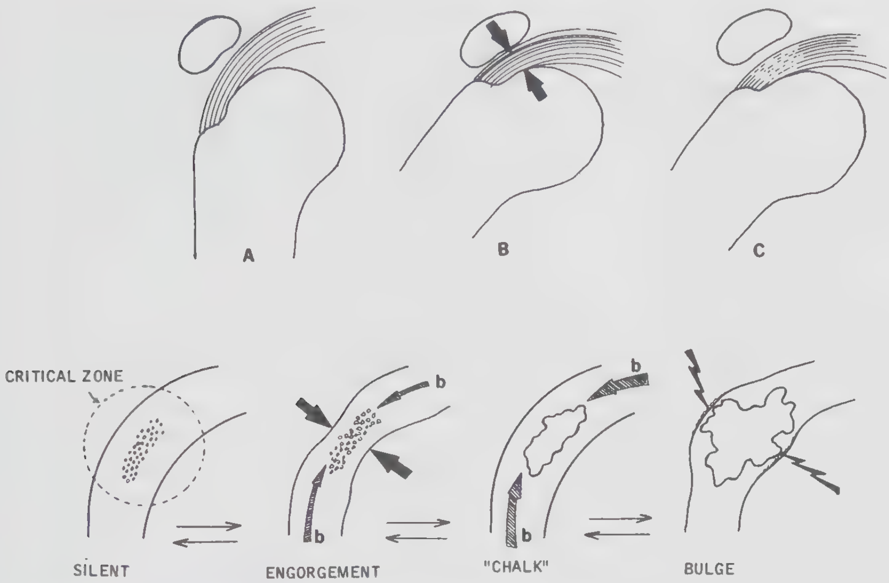


Figure 2–11. Natural sequence of calcific tendinitis. *(A)* the relationship of the supraspinatus (cuff) tendon between the coracoacromial ligament and the head of the humerus. *(B)* The repetitive pressure from daily use and abuse. *(C)* The degenerative changes of the tendon in the critical zone. The lower sequence follows the silent asymptomatic phase in the sequence steps to that of symptomatic *bulging*. Compression from whatever external cause results in engorgement through the tendon circulation. The debris of the *silent* phase absorbs fluid, and the dry powder becomes chalk. Further irritation and engorgement cause the chalk to expand, or bulge. The double arrows signify that each phase is reversible.

It has been claimed that x-ray studies of the shoulder reveal an incidence of shoulder calcium in 8 percent of the population over the age of 30 (Boyle), and so it becomes apparent that 8 percent of the population are candidates for calcific tendinitis with relatively minor trauma. Codman postulated that any calcific deposit greater than 1.5 cm in diameter would become symptomatic and that 35 percent of any calcific deposit would become symptomatic in time.

The bulge phase of the calcium deposit presents a mechanical obstacle to abduction and overhead elevation. The painful arc is again manifested as the arm reaches 60° to 70° of abduction. Repeated abduction and overhead elevation increases the inflammation and thus increases the amount of dissolved contained calcium.

Calcific tendinitis may present as a chronic aching in the shoulder aggravated by motions of forward flexion, abduction, or external rotation with or without overhead elevation. This chronic aching and aggravation prohibits many activities of daily living and participation in athletic events.

A more common presentation in the acute calcific tendinitis or exacerbated chronic calcific tendinitis is excruciating shoulder pain that comes on suddenly and immediately prohibits any movement of the shoulder without excruciating pain. The pain may radiate from the local suprahumeral space to the insertion of the deltoid muscle, the lower aspect of the upper third of the outer humerus. The patient not only cannot move the arm but also cannot sleep, cannot find a comfortable position, cannot bear any local pressure, and must avoid almost *all* arm motion.

The pathologic process here is that of a local *calcium boil* causing irritation and pressure upon all the contiguous tissues: the supraspinatus tendon, the subdeltoid bursa, the inner upper synovial capsule, and even the biceps tendon.

When in the supraspinatus tendon, calcium deposits responsible for the acute symptoms are aggravated by forward flexion, abduction, and some external rotation. When there is a calcium deposit in the infraspinatus and the teres minor muscle, the pain is noted posteriorly during shoulder rotation. Calcific deposits in the subscapularis muscle and its tendon are usually asymptomatic.

In the acute episode any and all motions of the shoulder are avoided. There is a region of maximal tenderness, and frequently there is local fullness and erythema over the area.

Rest of the part and modalities such as ice packs, deep heat, ultrasound, and oral anti-inflammatory medication may allow the inflammation to subside and the chalk to decrease in volume. It may resort to a smaller, drier, chalkier silent phase.

The calcium boil may spontaneously rupture into adjacent (Fig. 2–12) tissue, immediately relieving acute pain. If the patient can await this development, it is the natural progression of the condition. If the pain is too excruciating to accept, a surgical release of the boil with or without aspiration may be needed.

Aspiration of this calcific accumulation within the tendon may require merely a simple injection into the deposit with a fairly large-bore needle. An anesthetic agent in the solution, possibly including a steroid, may enhance the efficacy of the injection. Aspiration with *washing out of the calcium* has been advocated, in which two needles are inserted—one injecting and the other aspirating—but the benefit gained in this procedure is probably more the result of the decrease of tension within the tendon than of penetration of either needle.

Follow-up x-ray studies of the shoulder will usually reveal what has happened to the calcium deposit. The patient's relief, however, is the greater indication as to whether the tension within the boil has been released. In the patient who gets no relief from aspiration or needle insertion (estimated at 10 percent of those afflicted), direct incision through the deltoid muscle may be necessary.

Whatever the ultimate progression of the encapsulated calcium deposit, all the tissues adjacent to the calcium cyst remain inflamed, that is, the subdeltoid bursa, the biceps tendon, and even the glenohumeral capsule. Some residual inflammation that can lead to adhesive capsulitis and adhesive bursitis may remain that requires attention. This will be discussed subsequently.

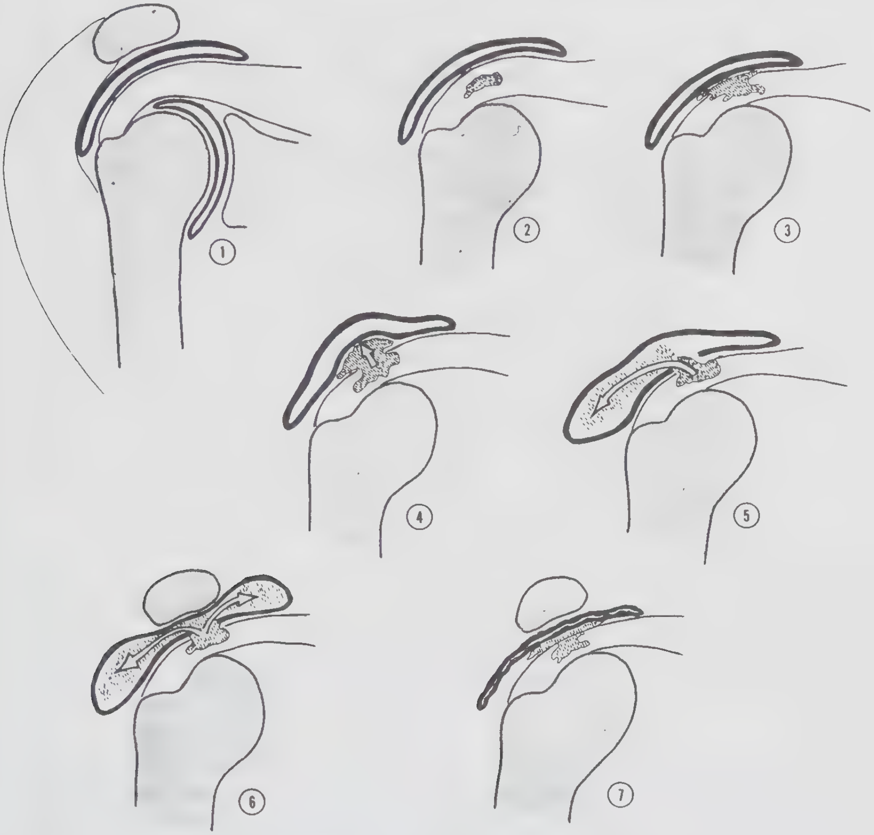


Figure 2–12. Evolution of the calcified tendinitis and formation of *bursitis*. *(1)* The normal relationship of the supraspinatus tendon (cuff) to the coracoacromial arch and the head of the humerus; the intimate relationship of the subdeltoid bursa and the glenohumeral joint. *(2)* The site of calcium deposit in the cuff tendon. *(3)* The *bulging* calcium has been evacuated from the tendon into the subbursal space. *(4)* A partial evacuation into the subbursal space with much debris remaining within the tendon. *(5)* Tendon evacuates, with rupture into the subdeltoid bursa. *(6)* *Dumbbell* intrabursal invasion. *(7)* Adhesive bursitis in which there is thickening of the walls of the bursa and adhesion between the superior and inferior surfaces.

BURSITIS: SUBDELTOID (SUBACROMIAL) BURSITIS

The condition previously discussed under the term supraspinatus tendinitis is commonly termed *bursitis*. A primary subdeltoid bursitis is a rare occurrence. Usually the bursitis is a concomitant inflammation of the adjacent tendinitis. Anatomically the inner synovial wall of the subdeltoid bursa is the outer

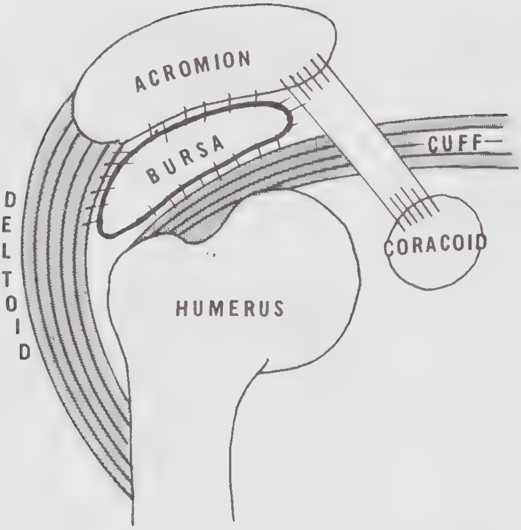


Figure 2–13. Subacromial bursa. The subdeltoid bursa is essentially the inner synovial lining of the deltoid muscle and the undersurface of the acromium. The outer layer of the supraspinatus portion of the cuff is the inner layer of the bursa. In any movement of the arm, the two layers of the bursa glide upon each other as the bursa deforms.

wall of the supraspinatus tendon (Fig. 2–13). One cannot be separated from the other, and thus when the outer layer of the tendon sheath is inflamed, so is the inner wall of the subdeltoid bursa (Fig. 2–14).

A primary subacromial bursitis can occur, but aspiration of the bursa will reveal its origin and the offending agent. In general rheumatoid disease, a primary bursitis can result, as can an acute bursitis, from direct trauma.

Diagnosis: Supraspinatus Tendinitis (Subdeltoid Bursitis)

Many of the diagnostic aspects of supraspinatus tendinitis have been presented here, but a summary is warranted. As stated earlier, the history may be unrevealing as to when, how, and why the tendon has been irritated, traumatized, or inflamed.

The adage regarding the cause of most musculoskeletal pain syndromes applies: *Pain can result in any of three conditions (1) abnormal strain on a normal joint, (2) normal strain on an abnormal joint, or (3) normal stress on a normal joint when the joint is unprepared or improperly executed for the performance of that particular activity.*

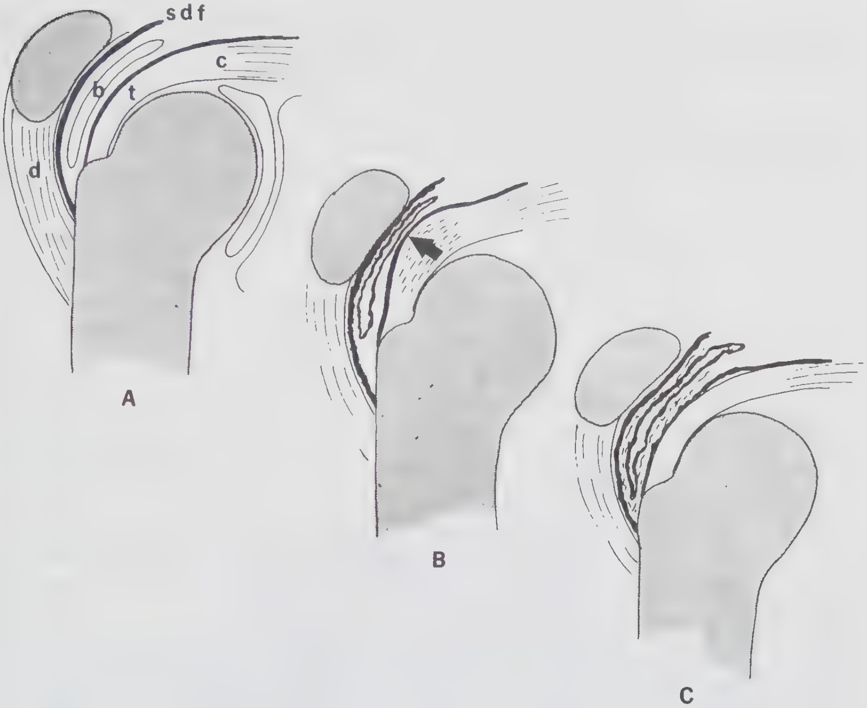


Figure 2–14. Acute tendinitis and inflammation of contiguous tissues. *(A)* The tissues contained between the rotator cuff (c) and its tendons (t), and the subdeltoid fascia (sdf) lining the undersurface of the deltoid muscle (d). The subdeltoid fascia is rich in blood vessels and sympathetic nerves which originate in the stellate ganglia. Between the subdeltoid fascia and the fascia covering the cuff is loose connective tissue within which is located the subdeltoid bursa (b). *(B)* Acute bulging of the tendon compresses and causes inflammation and swelling of the fascial tissues and the bursa. This is the acute mechanical phase during which there is severe pain and mechanical limitation of motion. *(C)* The bulging of the tendon has subsided, but the resultant fascia and bursal inflammation persists, causing stiffness of the shoulder. *A* can go to *B* then to *C* and reverse through the entire phase back to *A.* The tendon remains frayed, and degenerative changes remain.

Abnormal stress (1) is evidenced in a direct blow or a fall upon the upper extremity. An abnormal joint (2) may be congenital, posttraumatic, or—even more subtly—one degenerated by repeated multiple minor traumas. The normal joint undergoing normal stress (3) from unpreparedness or improperly executed activity is the most common presenting condition. This implies that, for whatever reason, the arm violates the normal scapulohumeral rhythm; for example, abduction with improper external rotation, faulty scapular motion in coordination of humeral activities, faulty posture, and improper training for a specific (athletic or professional) activity. Faulty execution from fatigue, impatience, anger, or anxiety can become trauma-producing events. This last category (3) is essentially one of trauma, albeit less evident.

Pain is the initial symptom and varies in intensity and site. The intensity depends on the significance of the resultant inflammation or tissue damage to the tendon. The site is initially pointed to by the patient and elicited by the examiner. The sites are depicted in Figure 2–15.

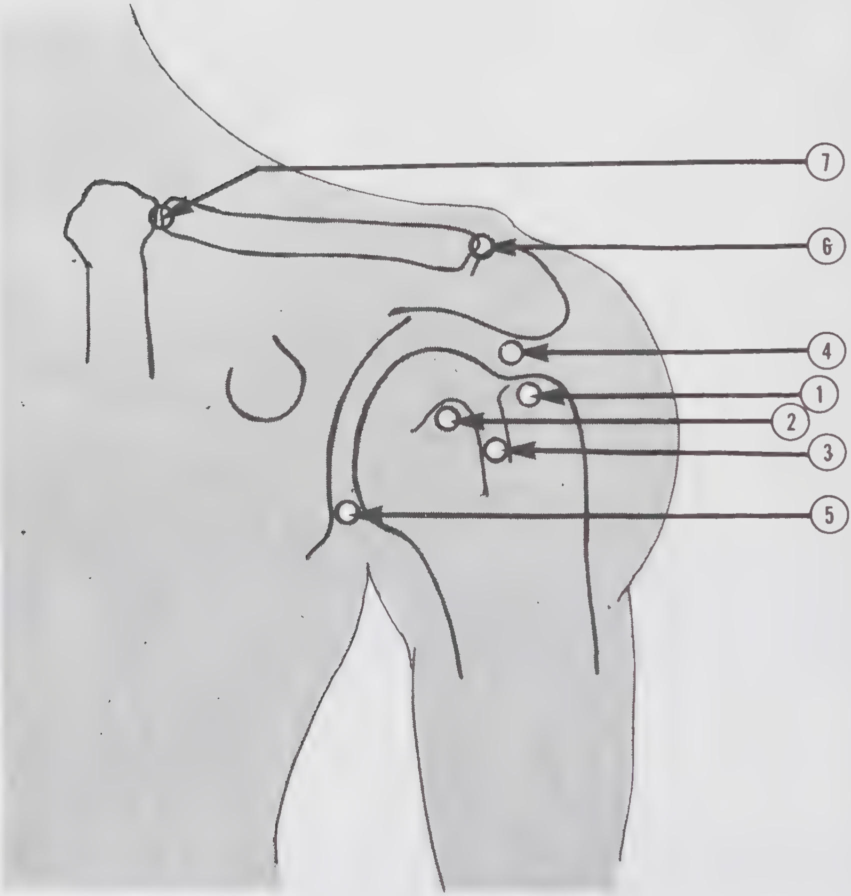


Figure 2–15. Trigger points. Palpable *trigger points* during the examination reveal the site of the pathologic process, corroborate the history, and indicate the type of therapy. *(1)* The greater tuberosity and the site of supraspinatus tendon insertion. *(2)* Lesser tuberosity, site of subscapularis muscle insertion. *(3)* Bicipital groove in which glides the bicipital tendon. *(4)* Site of the subdeltoid bursa. *(5)* Glenohumeral joint space. *(6)* Acromioclavicular joint. *(7)* Sternoclavicular joint.

Of greater diagnostic significance is the reproduction of or aggravation of pain from specific motions. Limited motion is protective, and which motion is limited is diagnostic (Fig. 2–16).

The patient often presents holding the arm supported in an elevated adducted and internally rotated position. This position decreases the tension (gravity) on the supraspinatus tendon, as does the internally adducted position.

Pain incurred upon a specific motion of the arm becomes diagnostic. Calcification of the supraspinatus tendon usually causes pain on forward flexion or abduction of the arm. Calcific deposits in the infraspinatus and teres minor muscles usually cause pain on external rotation of the humerus. Calcific deposits within the subscapularis muscle are usually asymptomatic.

Abduction is avoided either actively or passively because this further entraps the supraspinatus tendon against the overhanging acromium and coracoacromial ligament. In the patient with a rotator cuff inflammation and/or calcific tendinitis, the attempt at abduction and overhead elevation occurs as a *shrugging mechanism.*

Shrugging Mechanism. In attempting abduction of the arm, the patient exhibits the classic shrugging mechanism. This is significantly diagnostic as to merit emphasis. Normally the humerus abducts and rotates externally with concomitant synchronous scapular rotation and elevation. When the glenohumeral motion is limited or prohibited due to supraspinatus tendon engorgement, any motion attempting abduction and arm elevation occurs at the scapula.

The scapula thus elevates and rotates, whereas the humerus does not abduct or rotate externally (Fig. 2–17). This shrugging is an active motion requested of the patient. If the arm can be passively elevated (abducted) past the point of entrapment (by the examiner), further abduction elevation may be possible. Pain recurs upon active descent of the arm toward the side position. This pain occurring at a specific point of abduction and again at the same point of descent is known as the painful arc (Fig. 2–18).

As the inflamed tendon passes by sites of potential entrapment in external-internal rotation as well as in abduction, a painful arc during external-internal rotation is also possible, but this is not as apparent nor as frequently present.

The shrugging mechanism is emphasized in diagnosing supraspinatus tendinitis, but, as will be detailed, it is also very diagnostic in delineating the presence of complete or partial rotator cuff tears. Also, in the presence of adhesive capsulitis and adhesive subdeltoid bursitis, the shrugging mechanism is present for the same reason, that is, limitation of glenohumeral motion and free scapulocostal motion.

On examination there is usually a local fullness or swelling about the area of the local tenderness.

Radiologic Diagnostic Tests. In a calcific tendinitis there are often rounded deposits of haziness found between the acromium and the greater

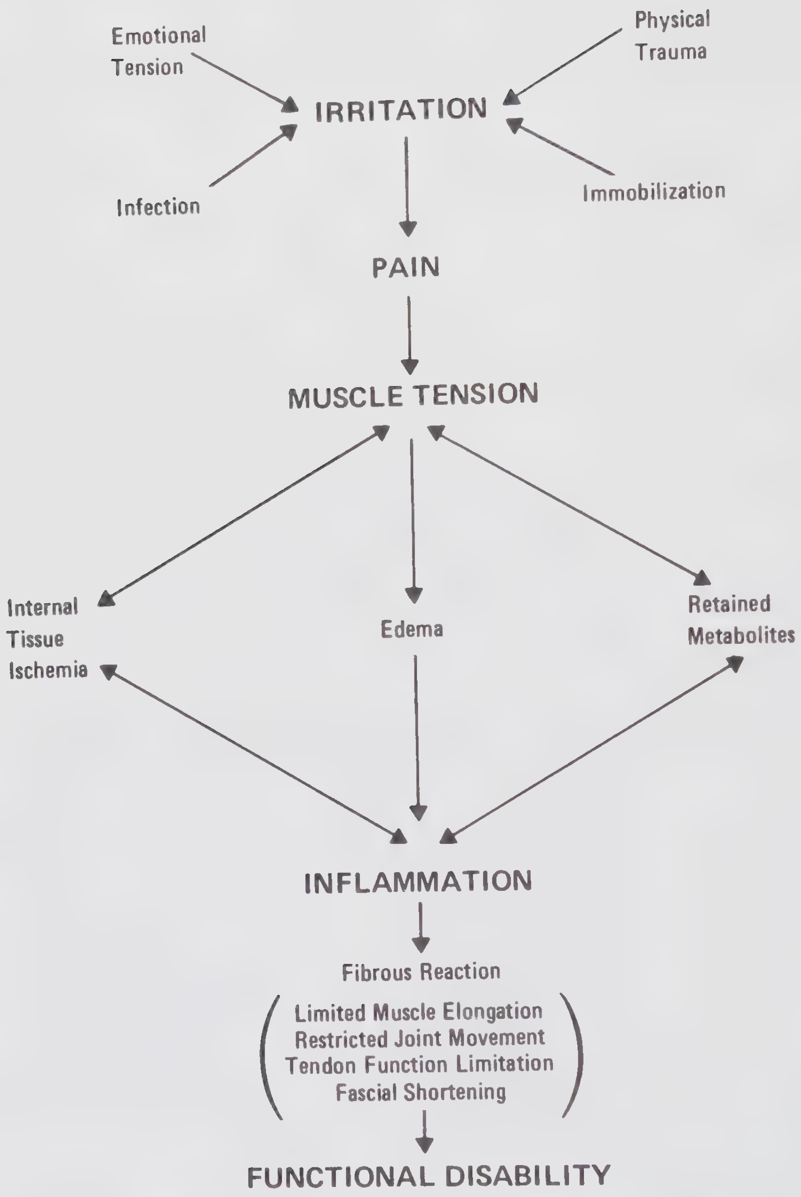

Emotional Tension
Physical Trauma
IRRITATION
Infection
Immobilization
PAIN
MUSCLE TENSION
Internal Tissue Ischemia
Edema
Retained Metabolites
INFLAMMATION
Fibrous Reaction
Limited Muscle Elongation
Restricted Joint Movement
Tendon Function Limitation
Fascial Shortening
FUNCTIONAL DISABILITY

Figure 2–16. Mechanism by which irritation leads to functional disability.

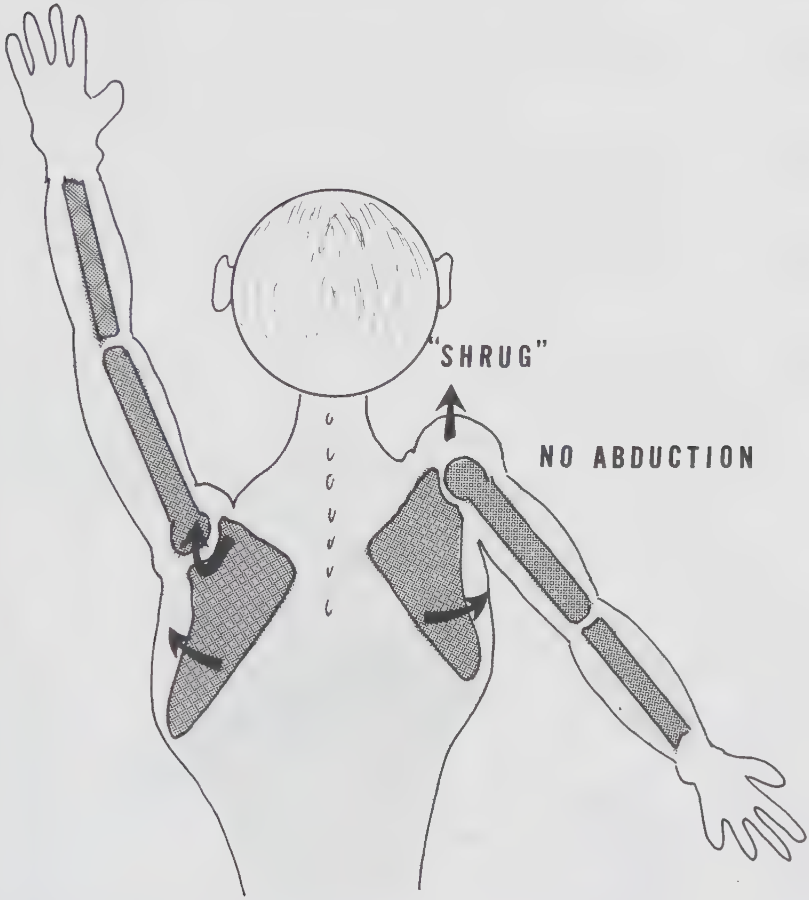


Figure 2–17. Shrugging mechanism. As pictured on the right side, the glenohumeral joint allows no abduction and limited external rotation due to either calcific tendinitis, supraspinatus tendinitis, rotator cuff tear, or an adhesive capsulitis. In the attempt to abduct the arm *only*, the scapular aspect of the glenohumerus rhythm functions and the scapula *shrugs,* and there is no movement at the glenohumeral joint.

tuberosity of the humerus. Several views of the arm may need to be taken to reveal these deposits. Without visible calcific deposits, routine roentgenograms of the average tendinitis may be nonrevealing.

Treatment

Initially, during the acute phase, rest of the part is desirable. The part here is the glenohumeral joint. Local ice packs applied for 20-minute periods three

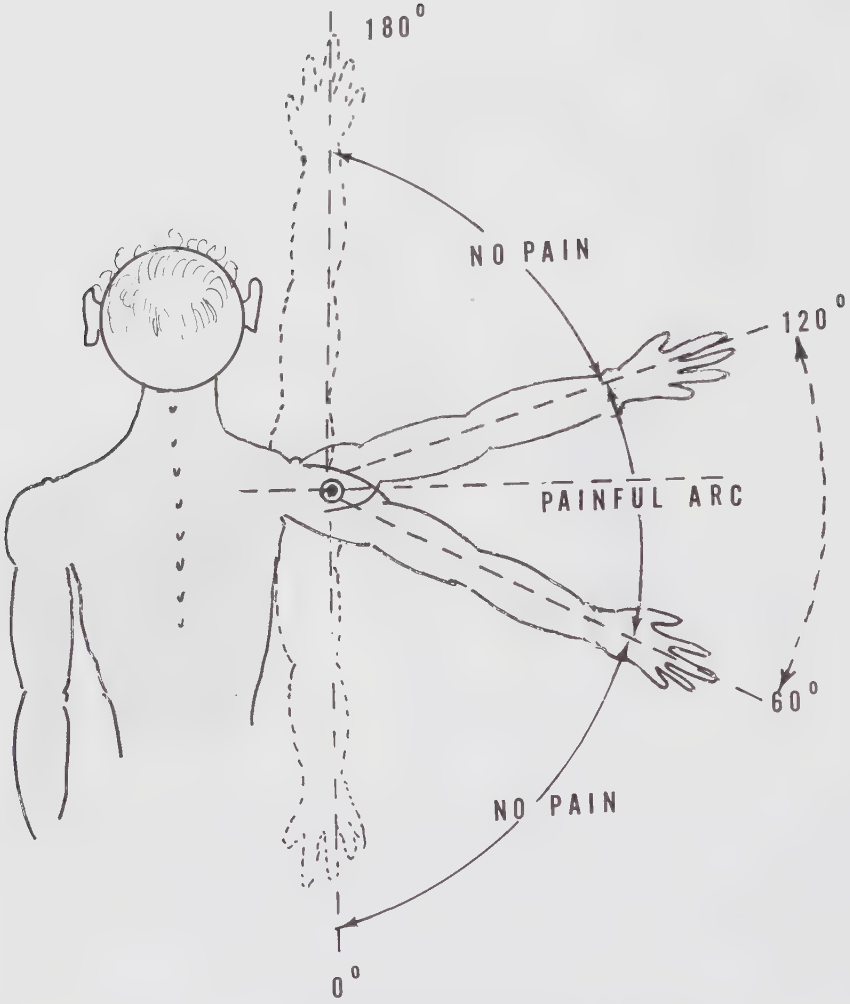


Figure 2–18. Painful arc. In a supraspinatus tendinitis there is free painless motion of abduction from dependency (0°) to 60°. From 60° to 120° there is pain, both on elevation and descent. From 120° to overhead elevation 180° the motion is pain free. It is between 60° and 120° of abduction (and between 120° and 60° of descent) that the greater tuberosity and thus the conjoined tendon insertion passes under the acromium and the coracoacromial ligament.

to five times daily is valuable. It has been shown that intramuscular temperatures at a depth of 3 cm are not affected until after 10 minutes of a cooling period.

Ice is analgesic, it decreases further inflammatory chemical and vascular changes, and it minimizes protective *spasm.* Heat initially may be soothing, but it also may enhance further engorgement of these confined tissues. After 48 hours, heat enhances the healing process, removes debris and chemical toxins, and brings a new vascular supply to the injured part. Heat also has been shown to prevent fibrillar collagen intersection adhesion (Kottke) in capsules (Fig. 2–19).

A sling to elevate the arm with the elbow flexed and to position the arm in internal rotation may be of considerable comfort, but a sling and thus immobilization must never be prolonged. An arbitrary time of use for a sling may be 48 to 72 hours, with gradual removal and implementing of motion of the glenohumeral joint, first passively and then actively.

Immobilization fosters tissue ischemia and an accumulation of the noci-

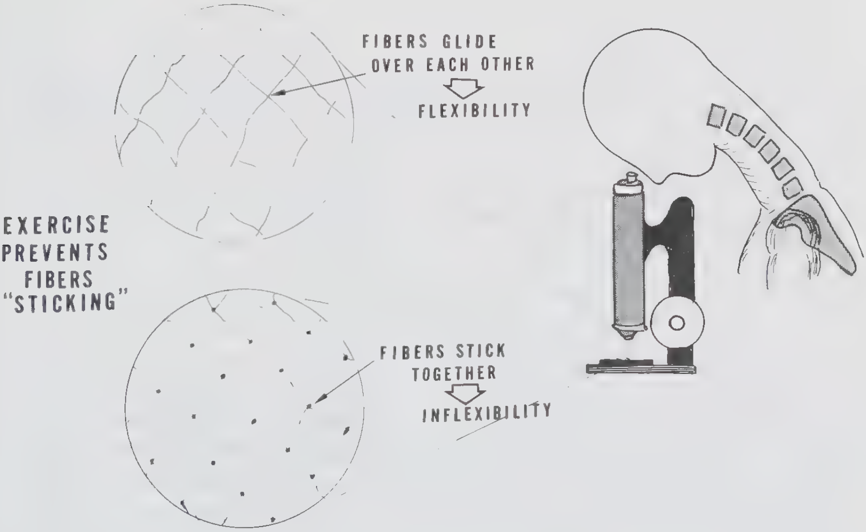


Figure 2–19. Collagen fiber intersectional gliding. In the above microscopic field the collagen fibers of a capsule crisscross and at their intersection they glide over each other. The lubrication for this is from the matrix and repeated motion.

In the lower field the fibers have bound at their intersection, causing inflexibility. The elongation of each fiber is now between points of adhesion rather than elasticity of the entire collagen fiber. (From Cailliet, R and Gross, L: The Rejuvenation Strategy. Doubleday, New York, 1988.)

ceptor metabolites. The edema that results from trauma tends to organize, and the synovial linings of the bursa and capsular layers become inflamed and thus adhesive. Allowing prolonged intimate contact of these synovial layers encourages adhesive capsulitis and adhesive bursitis (Neviaser, 1945; Watson-Jones) (Fig. 2–16).

To prevent adhesive reaction, passive rather than active motion must be initiated early. Paradoxically, passive exercises should be done actively by the patient. These passive range of motion exercises done actively by the patient are called *Codman exercises.*

The reason for passive range of motion is to ensure frequent movement of adjacent synovial layers and to avoid prolonged contact and thus adhesion of the adjacent inflamed surfaces. The reason for avoiding active motion is that active motion requires contraction of the rotator cuff muscles, which initiates irritating contraction of the rotators and abductors with compression against the overhanging acromium and coracoacromial ligament.

The Codman exercise is essentially a *pendular exercise.* The person bends forward at the waist with the affected arm dangling vertically (Fig. 2–20). This places the arm at 90 degrees of forward flexion without any deltoid or rotator cuff muscular contraction. Gravity on the dependent arm causes a slight separation of the glenohumeral surfaces. The glenohumeral capsule is also consequently slightly elongated.

The body then must be moved forward, backward, and in a circumducting manner with the arm *passively* moving. The arm is a passive pendulum, being moved in all directions of glenohumeral motion without active glenohumeral muscular contraction.

Active pendular exercise (Fig. 2–21) utilized later to regain muscular strength is to be avoided initially in treatment. This active pendular exercise encourages active contraction of all the glenohumeral muscles, defeating the concept of passive pendular exercise. At a later stage of rehabilitation this active pendular exercise is effective and desirable, but only *after* pain has significantly subsided and motion is possible with less discomfort.

It may seem redundant, but experience mandates that it be stated. The passive pendular exercise must be done properly to be effective. This means a careful explanation to the patient and observation by the therapist. Done improperly, this exercise not only fails to benefit but also may be detrimental.

Bending forward at the waist must be done with flexed knees to avoid low back discomfort. A dependent arm means that there must not be any muscular effort. This indicates total relaxation of the entire shoulder musculature and thus a dependency on the capsule toward which this exercise aims. Movement forward, backward, sideways, and circumduction must be done *with the body, not the arm.* The arm merely becomes pendular as the body moves.

There is a natural tendency for the patient to swing the arm in all directions, but if this becomes an *active* muscular exercise, it defeats the passive pendular action. In a later phase of treatment the pendular exercise must be used when

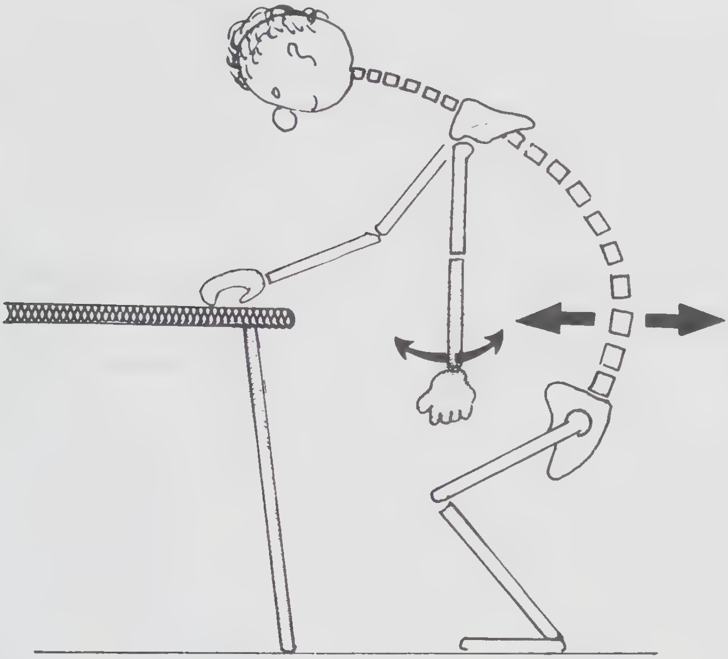


Figure 2–20. Codman pendular exercise. The patient bends forward, flexing the trunk to right angles. The involved arm is dangled without muscular activity of the glenohumeral joint. The body actively sways, thus passively swinging the dependent arm in forward flexion-extension, lateral swing, and rotation. The body can be supported by placing the other arm upon a table or chair. The arm is *passively* swung. No weight is held in the hand because this causes muscular contraction of the arm and the shoulder.

it becomes necessary to begin active exercises to strengthen the shoulder muscles.

Adding a weight to the hand to enhance the effectiveness of the pendular exercise is counterproductive. Manual grasp of the weight automatically causes cocontraction of the arm and the shoulder muscles. In the passive pendular exercise this is undesired. A weight in the hand will be acceptable and even desirable when the pendular exercise progresses to the active rehabilitation phase.

Heat can be applied before and during the pendular exercise, as can ice. Whether to apply ice or heat will be dictated by the stage (acute, subacute, or chronic) of the condition, the severity of the pain, and the intent of the exercise—increase in range of motion or strengthening of the muscles.

The passive pendular exercise is the first stage in recovering full range of motion, which means regaining full flexibility of the capsule of the glenohumeral joint. Failure to regain full flexibility denies gliding of the humeral head on the glenoid fossa, which thus impairs full scapulohumeral rhythm. Aspects of

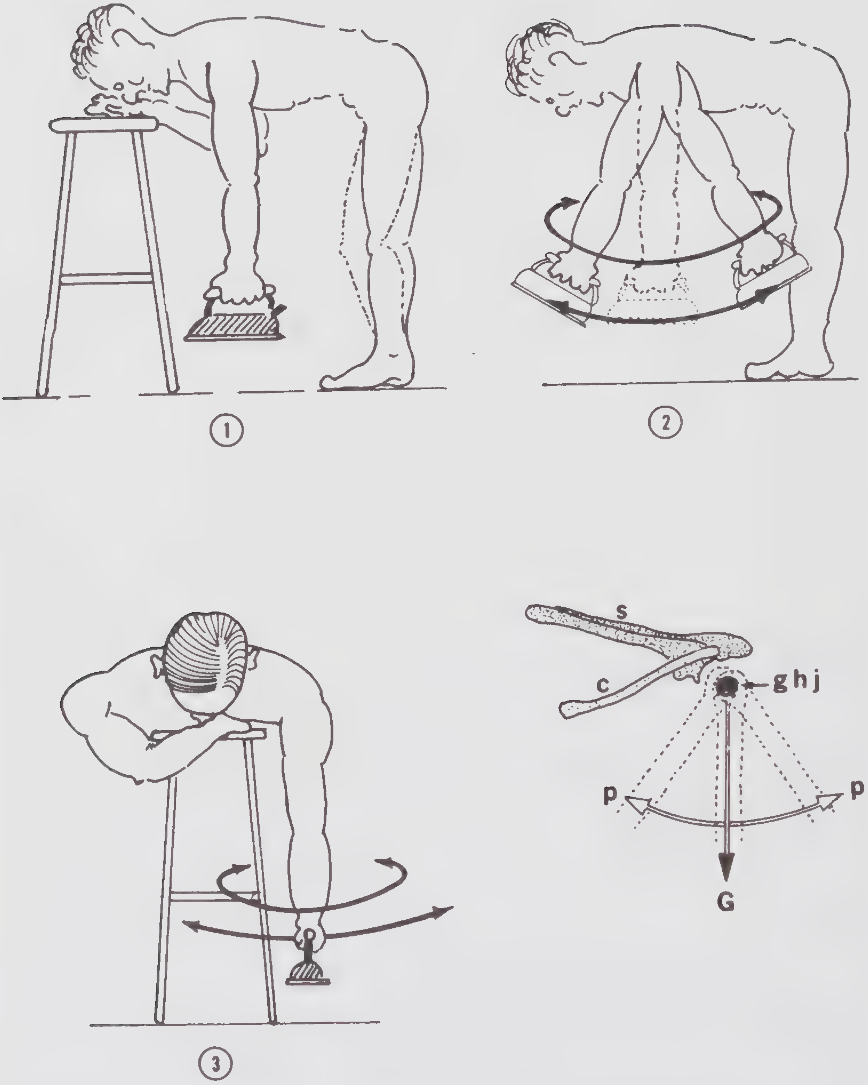


Figure 2–21. Active pendular glenohumeral exercise. *(1)* The posture to be assumed to permit the arm to dangle freely, with or without a weight. *(2)* The arm moves in forward and backward sagittal plane, in forward and backward flexion. Circular motion in the clockwise and counterclockwise direction is also done in ever-increasing large circles. *(3)* The front view of the exercise showing lateral pendular movement actually in the coronal plane. The lower right diagram shows the effect of gravity (g) upon the glenohumeral joint (ghj) with an immobile scapula (s). The p-to-p arc is the pendular movement.

this limitation will constitute varying degrees of *frozen shoulder*, from subtle signs of adhesive capsulitis to a complete adhesion. This condition will be discussed later, but it must be mentioned here in the early stage to justify the indication for early passive then active range of motion exercises.

Because subdeltoid bursitis and supraspinatus tendinitis indicate inflammation of the rotator cuff and its contiguous tissues within a confined space, every attempt to combat inflammation is indicated. An injured tissue initiates a release of arachidonic acid, which is followed by an ultimate release of other chemical compounds: prostaglandins, thromboxane, monohydroxy fatty acids, and leukotrienes. These leukotrienes are considered the agent that gives rise to inflammation. Hyperalgesia results. A subsequent chemical reaction then results with release of bradykinin and histamine, which causes pain, edema, and vasomotor reaction.

This is taken under consideration by the use of local application of ice and by resting the part. Oral anti-inflammatory drugs are very important in disrupting this chemical cycle of inflammation and its sequelae. Aspirinlike medications—nonsteroid anti-inflammatory drugs (NSAIDs)—disrupt the initial arachidonic acid formation into prostaglandins. Ice also diminishes the action of the histaminelike effects. Oral medication is effective but requires careful monitoring for avoiding other medical contraindications. Oral steroids are even advocated in a severe acute condition that does not respond to aspirin or NSAIDs.

Local injections of anesthetic agents combined with steroids into the suprahumeral space that surrounds the supraspinatus tendon, its sheath, the bursal synovium, and glenohumeral capsule synovium intervene in the cycle of nociceptor chemicals, prevent accumulations of these kinins, decrease pain by decreasing local inflammation, and prevent formation of adhesive factors of the synovial tissues.

The technique of injection may vary, but its value now has been confirmed. A word about technique, possibly superfluous, is worthwhile: The region to be penetrated is reasonably large and devoid of major nerves and blood vessels. The finger of the examiner—not the needle—must determine the site of entry of the needle. Once past the skin and under the overhanging acromium (which is palpable by the finger of the physician) the needle permits simple entry to the suprahumeral space. Aspiration to avoid entry to a blood vessel is mandatory, as in any injection. The posterior entry is preferred by many (Fig. 2–22).

The spine of the scapula is palpated laterally to its end, which is the acromial process. Several centimeters medially and directly under the lower edge of the acromium is the site of entry of the needle. Entry for a few centimeters in an upward direction reaches the desired suprahumeral area.

The lateral entry requires palpating the outer inferior edge of the acromium and entering immediately below that edge. After a direct entry followed by a progression of 1 to 2 cm in a slightly upward (inward) direction, the desired space is reached.

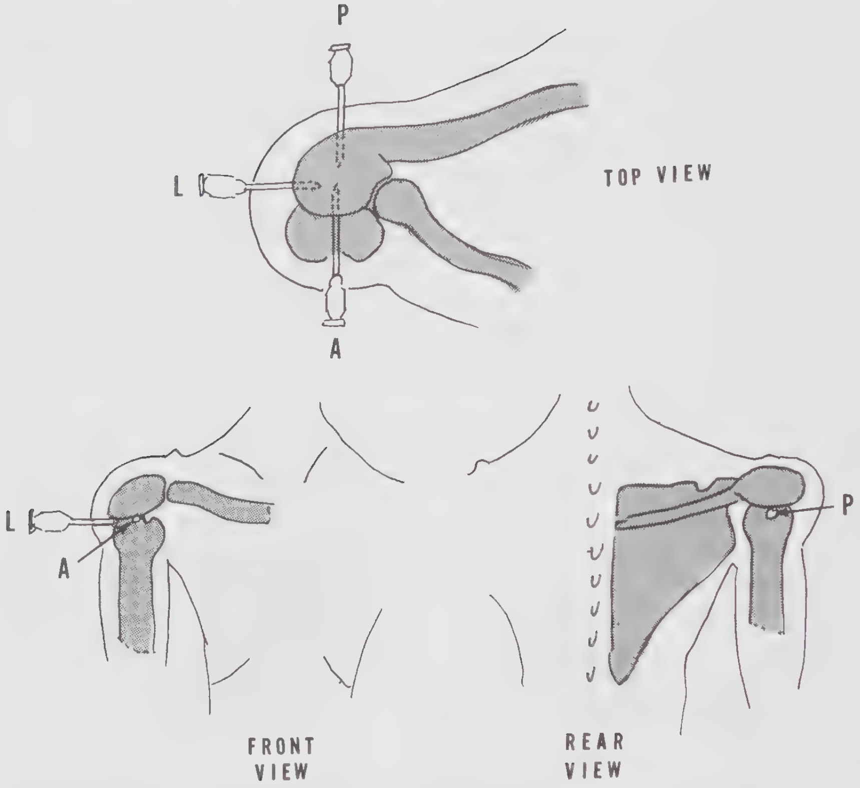


Figure 2–22. Sites of suprahumeral injections. In injection therapy of supraspinatus tendinitis an injection of an anesthetic agent and a steroid solution can be made from three approaches. The solution is ultimately to be deposited in the suprahumeral space around the conjoined tendon.

A, anterior approach: Under the inferior edge of the overhanging acromium and slightly lateral to the bicipital groove. All these landmarks are directly palpable. L, lateral approach: Directly lateral and immediately below the inferior lateral margin of the acromium. P, posterior approach: Following the scapular spine laterally the inferior margin of the acromial process is palpable, and the entry is directly under that margin, proceeding anteriorly and slightly upward.

The anterior entry requires palpating the space immediately under the inferior anterior edge of the acromial process, then palpating for the bicipital groove. Once palpated, the bicipital groove can be verified because it moves as the humerus rotates slightly in an external and internal direction. The reason for palpating the bicipital groove is to locate the lateral tuberosity upon which inserts the conjoined tendon. Entry of the needle is immediately under the anterior inferior edge of the acromium, over the greater tuberosity, which is immediately lateral to the bicipital groove. The needle then proceeds inward about 1 to 2 cm, then slightly upward for another 1 to 2 cm. Experiencing bony resistance immediately after penetrating the skin indicates entry into the wrong site. The needle must be withdrawn and reinserted through the same skin site of entry. As in any injection, aspiration must always precede injection of the fluid.

When pain persists in spite of suprahumeral anesthetic steroid injection and/or oral anti-inflammatory nonsteroidal medication, a suprascapular nerve block may be effective in affording relief (Fig. 2–23).

The technique for injecting the suprascapular nerve is as follows: With the patient seated and the arms at the side, the spine of the scapula is palpated. The vertebral (medial) endpoint of the spine of the scapula is marked, as is the acromial end of the spine. The distance between the two is bisected, and the outer half is again bisected. This is the site of emergence of the fossa through which the nerve passes. A wheel is injected under the skin approximately 1.5 cm above the superior edge of the spine. This is the point of entry.

A 24-gauge 2-inch needle, connected to a 10-ml syringe containing 1 percent lidocaine and possibly 1 ml soluble steroid, is inserted directly. Insertion proceeds slowly until bone is contacted, then is withdrawn a small fraction of a centimeter. After aspiration, the solution is slowly injected. Should there be failure to contact bone after several centimeters of penetration, the possibility exists that the fossa has been entered and further penetration could be into the pulmonary area. Immediate withdrawal is indicated.

Upon initial entry, if the suprascapular nerve is contacted, indicating that the suprascapular fossa has been entered, paresthesia is immediately noted by the patient. The needle immediately should be withdrawn a full centimeter, leaving the needle still through the skin but not in the scapular groove. The needle then can be reinserted at a different angle and again slightly withdrawn upon contacting the bone. The injection now is initiated.

A successful block should afford relief of shoulder pain, albeit with possible transient weakness of the external rotators. The latter paresis recovers rapidly, and the pain relief persists for a period of several hours or more.

It is apparent that the intent is *not* to inject into the suprascapular nerve directly but, rather, to inject the solution into the space between the scapular surface under the supraspinatus muscle where it will disperse slowly to gradually invest the suprascapular nerve branches (Bonica).

Therapeutic Exercises

The *active-passive pendular exercises* initiate increased range of motion and thus aid in the regaining of capsular flexibility. Passive motion also increases the gliding movement between the synovial layers of the subdeltoid bursa and the synovial layers of the glenohumeral capsule adjacent to the peratenon of the rotator cuff. These tissues must remain lubricated and free to glide upon each

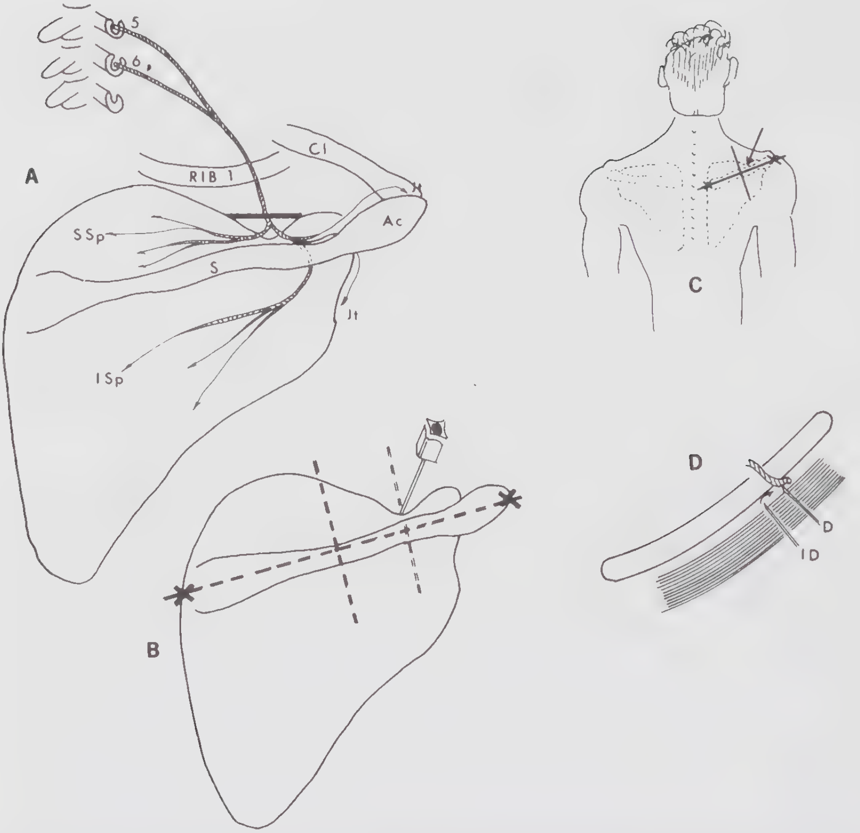


Figure 2–23. Suprascapular nerve block. *(A)* The anatomy and course of the suprascapular nerve, originating from C_{5-6}. The motor nerve to the supraspinatus (SSp), and infraspinatus (ISp) and the sensory branches to the acromioclavicular joint and the shoulder joint (jt) are shown. *(B,C)* Bisection of a line drawn along the scapular spine with the site of the groove and of needle insertion. *(D)* The difference between direct nerve contact and indirect (ID) by infusion along the posterior portion of the scapula below the supraspinatus muscle.

other for normal shoulder function.

After intracapsular and bursal anesthetic injections, the pain should subside sufficiently to allow more active exercises to proceed. The *passive* pendular exercise can now become *active* pendular exercise. Active pendular exercises indicate that the arm-shoulder motion is now preformed actively by the scapular glenohumeral muscles and not merely by body trunk motion. In addition to these active pendular exercises, other active exercises, such as those depicted in Figures 2–24 to 2–29, must be introduced.

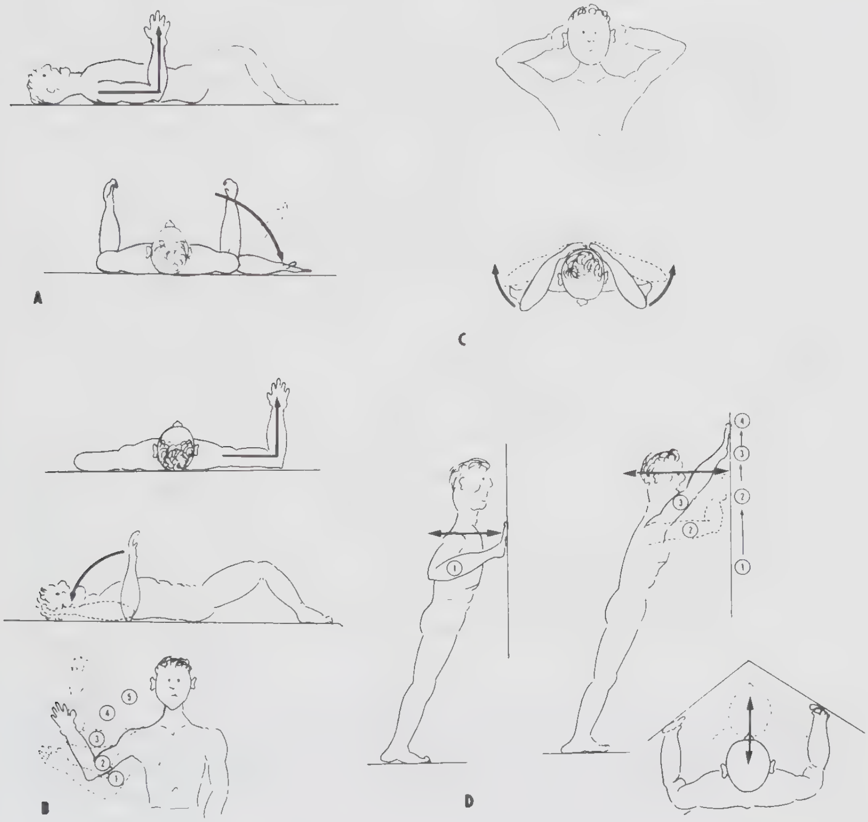


Figure 2–24. Exercises. *(A)* Lying on back with elbows at the side, hands toward the ceiling. External rotation is attempted actively by the patient and passively by the therapist. Resistance may be applied when range permits. This exercise can be performed in the upright position against the wall. *(B)* Similar exercise to A, but with increasing abduction of the arm through positions 1 to 5. *(C)* Hands behind head, the movement is backward motion of the elbows, to the floor when supine and to the wall when erect. This motion may be assisted by a therapist and may be resisted. *(D)* Push-ups from the wall performed in a corner. The exercise starts with hands at waist level; then the hands climb until they are fully extended overhead, still apart. The anterior capsule is stretched, as are the pectorals. Rhythm here is necessary. Avoid arching the back and the neck.

All active range of motion exercises must be done frequently (several times daily), gently but with some force to ensure gradual regaining of full range. Any exercise may be preceded by local heat application and followed by ice application, especially if there is some residual soreness.

It must be clearly stated that *full glenohumeral range of motion must be regained to ensure that normal scapulohumeral rhythm resumes.* Normal range

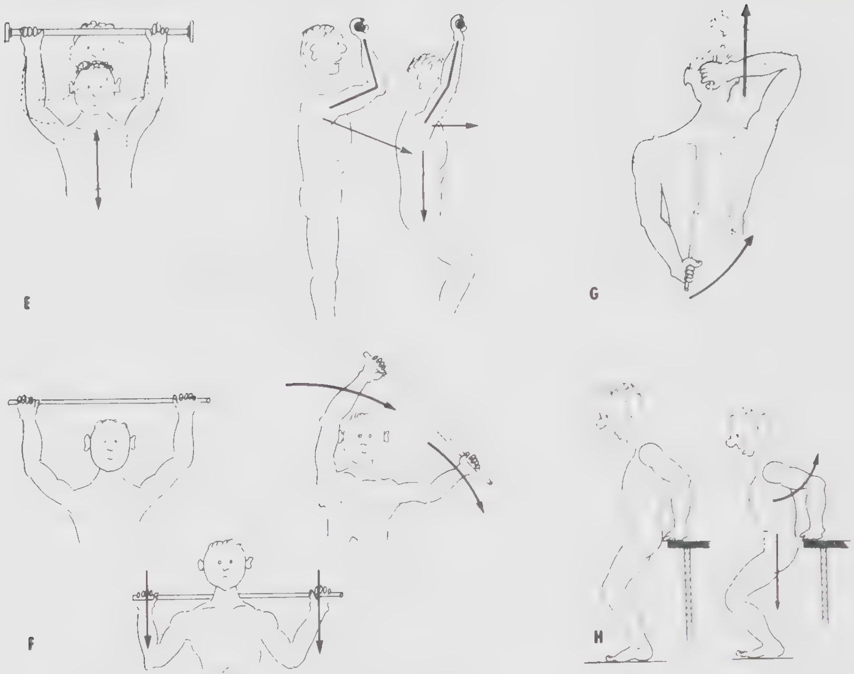


Figure 2–24 (*continued*). (*E*) With a chinning bar (between door frames) that is adjustable, begin with the bar at face level and gradually elevate the bar, either by changing the position in the door frame or by doing a deep knee bend. Ultimately the bar should be above and behind the head. (*F*) Similar to (*E*) except that a *wand* or wooden dowel is held by both hands. This exercise is more active than (*E*) the ultimate object being to place the wand behind the head from a fully extended overhead position. Lateral motion with the arms overhead should attempt movement of arms behind the head. (*G*) Wand behind the back. In the illustration the wand is elevated by the right hand to bring the left (involved) arm up behind the back, which stretches the anterior capsule and the external rotators. (*H*) Placing hands behind the back upon a table, parallel bars, or sink, and doing deep knee bends.

of motion must be compared with the contralateral shoulder joint. Remaining restriction, later to be discussed as subtle signs of adhesive capsulitis, may allow residual pain, impairment, and recurrence of further tendon degeneration and tearing.

Strengthening Exercises

In addition to full range of motion, strength and endurance of the rotator cuff muscles and of the other shoulder girdle muscles must be regained.

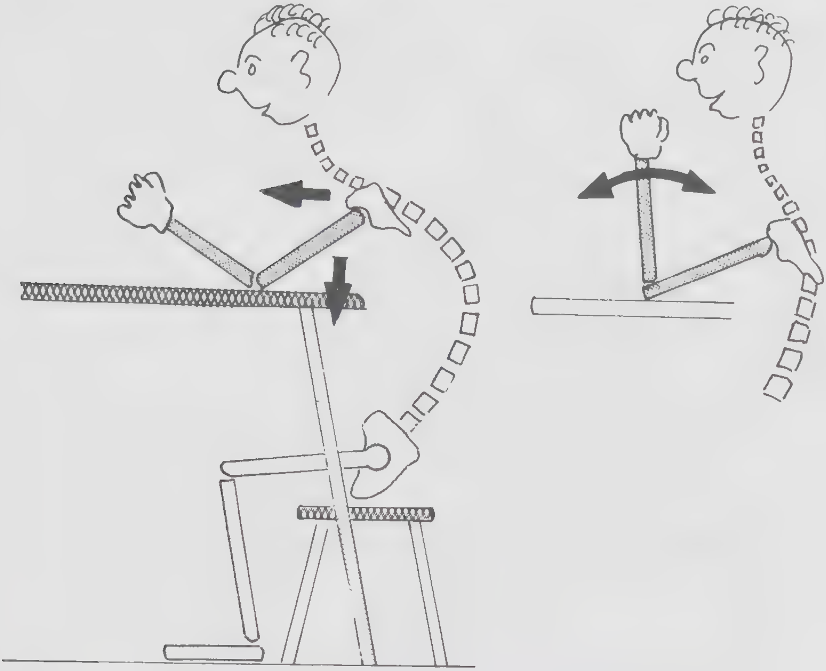


Figure 2–25. Home exercise to increase shoulder range of motion. Seated with arm supported upon table, the patient moves forward and downward to increase range of arm toward elevation. The forearm, bent at right angles, internally and externally rotates, thus further increasing range. A weight can be held in the hand.

Strength of the rotator cuff is stressed because shoulder-muscle tendon entity is the most important to be strengthened. Although tension and stretch have been incriminated in supraspinatus tendon degeneration and ultimate tear, gentle gradual repetitive tension applied to the tendon and its muscle has been confirmed to regain tensile strength of the tendon and to enhance repair. Further healing blood flow to the muscle tendon entity is enhanced by isometric and isokinetic strengthening exercises. Even partial tears have benefited by active exercises (this will be discussed in Chapter 3).

Because the supraspinatus muscle is primarily an external rotator, as well as the initiator of early abduction, external rotation is used to strengthen the muscle. Also, stressing external rotation avoids impingement when the exercise is done *without* abduction and overhead elevation.

Strengthening exercises of the rotator cuff muscles are exhibited in Fig. 2–30. With the arm at the side and elbow flexed to 90°, the hand goes from internal rotation to full external rotation without abducting the elbow from the side.

If the arm can be painlessly abducted to 90° of abduction, exercise to the external rotators can be performed in that position. With the arm in abduction,

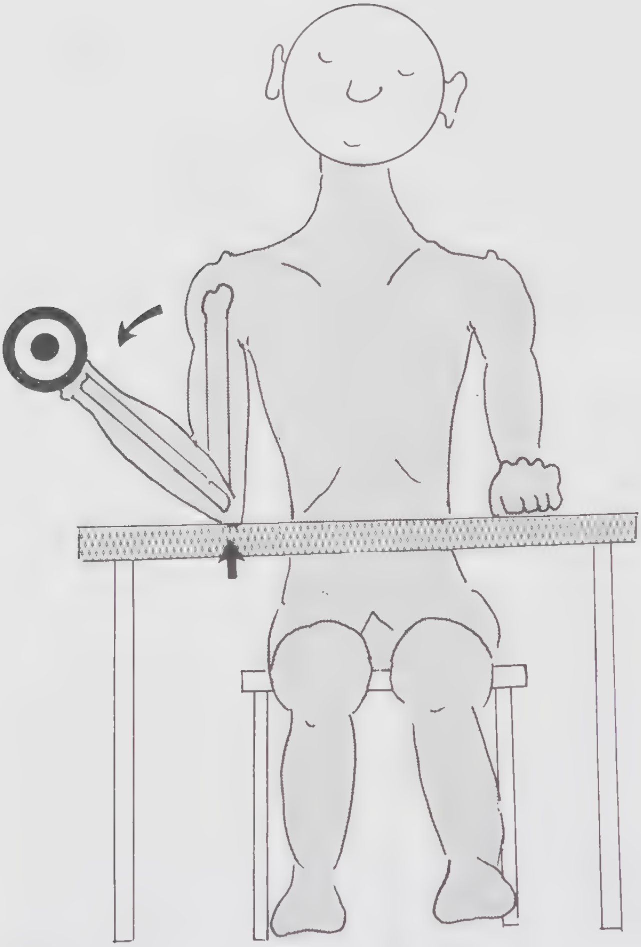


Figure 2–26. External range of motion exercise. While seated with the elbow bent to 90° of flexion, the person holds a weight in the hand and slowly and gradually drops the hand outward (externally rotating). The glenohumeral external rotation increases in range of motion. The capsule is gently and progressively stretched by this exercise. If done many times daily, it enhances the rapidity and completeness of regaining normal range of motion.

the flexed forearm can be rotated externally against resistance. During this latter exercise the upper arm may be supported passively to avoid excessive abduction activity of the rotator cuff and the deltoid.

Ultimately the deltoid muscle must also be strengthened, as must the shoulder scapular muscles. The ability to do these exercises without shoulder pain indicates that the supraspinatus tendon inflammation has significantly subsided.

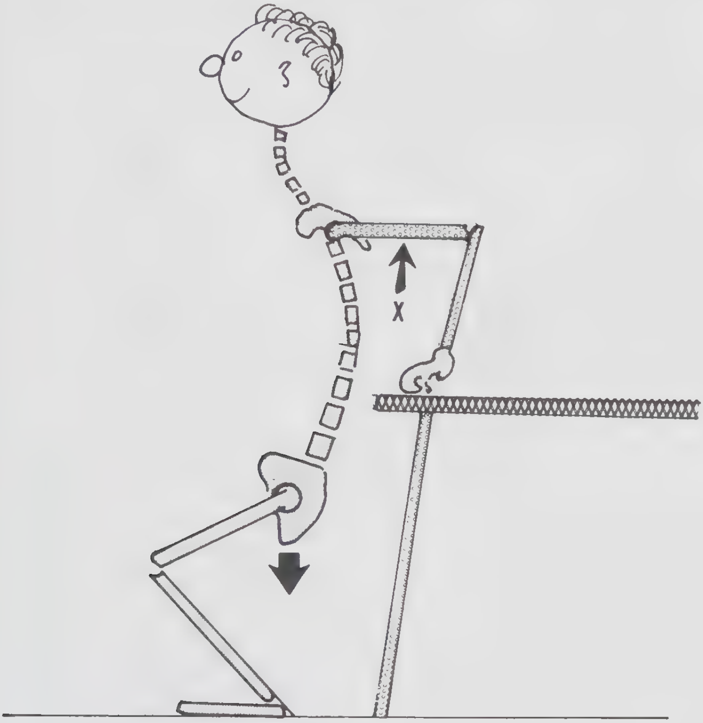


Figure 2–27. Exercise to stretch anterior capsule and to increase posterior flexion. The patient places both hands on a table behind the back and performs gentle deep knee bends. This elevates arms *(small arrow)* and increases posterior flexor range of motion.

An exercise that has been generally advocated, the *wall-climbing exercise* (Fig. 2–31), deserves mention because it may be beneficial in the recovery stage, but too often it is done improperly and so may be detrimental rather than beneficial.

Scapular mobility and strengthening exercises are beneficial because the scapular muscles have been shown to be weak in patients who develop myofascial scapular discomfort in occupational difficulties. If the scapular muscles are weak and the scapulothoracic flexibility is limited, it stands to reason that an additional burden on the glenohumeral muscle is imposed in activities of daily living or in athletic upper extremities activities (Fig. 2–32).

Physiologic Effect of Exercise

Exercise provides beneficial effects to all components of the musculoskeletal system—in this case, all the component tissues of the scapulohumero-

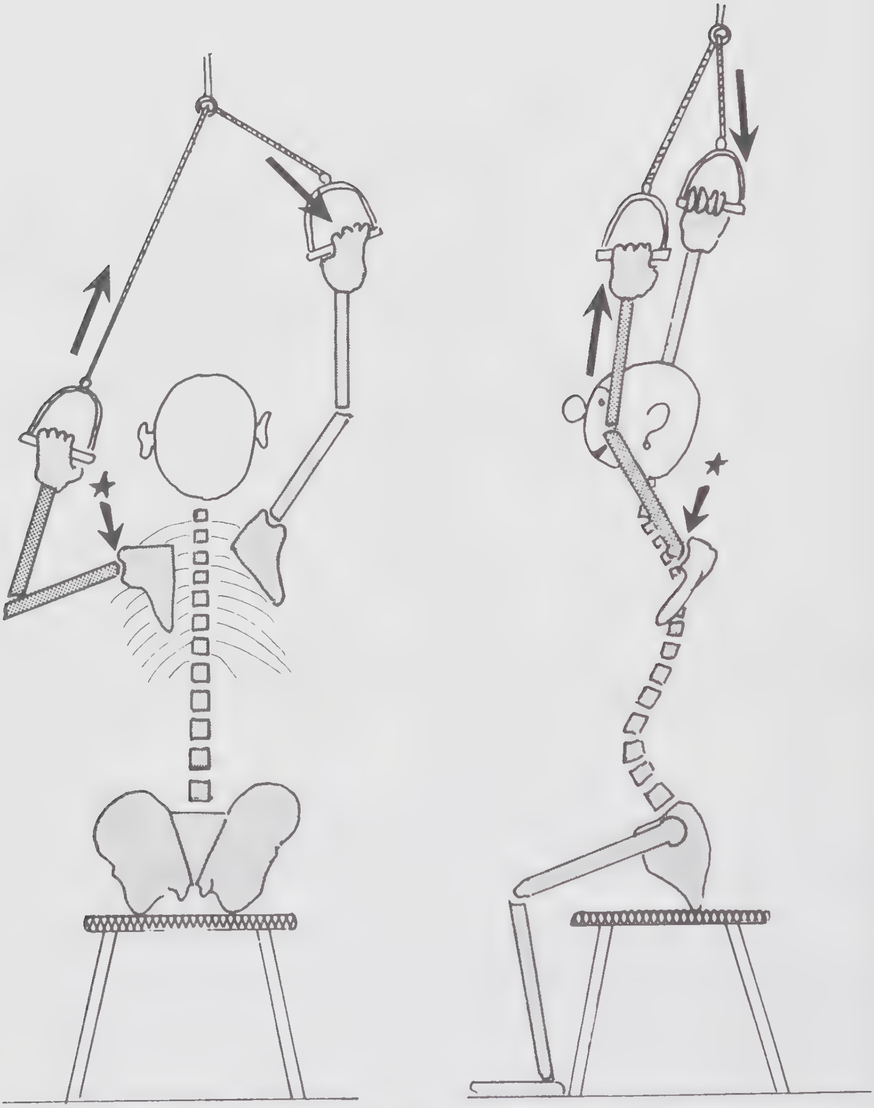


Figure 2–28. Overhead exercise. With a pulley placed above the head, the involved arm is passively elevated by the normal arm. By having the pulley slightly behind the head, the arm gets a farther range of motion to overcome one of the subtle signs of limitation.

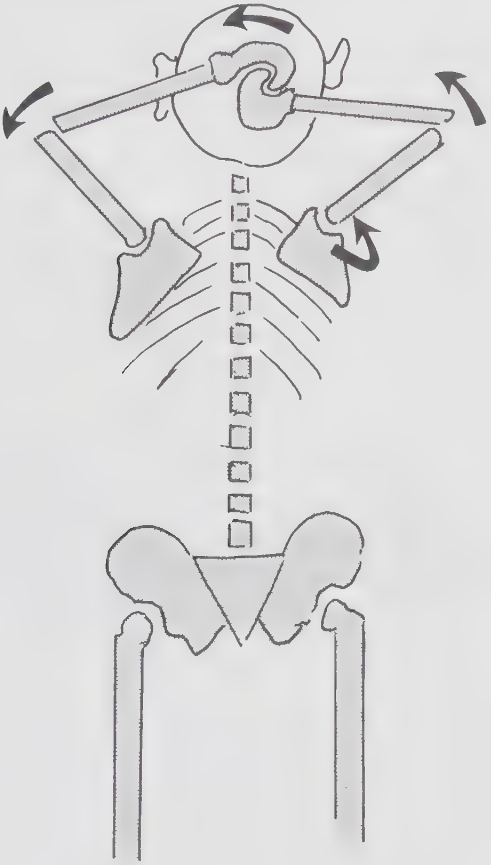


Figure 2–29. Overhead range of motion exercise. With the normal arm playing the role of the therapist, the hands are clasped overhead as far behind the head as possible. The normal arm pulls the limited arm over and behind the head. Gradually this increases the limited glenohumeral joint range of motion. The chance of joint capsule damage is minimized because the force is being applied by the patient. Inasmuch as it is a home exercise, it can and should be done four to ten times daily.

thoracic entity. Repetition of motions, so often advocated as an exercise benefit, is not necessarily beneficial to the glenohumeral joint. Especially if there is a painful arc, which indicates mechanical stress, as each rotation-abduction entraps the inflamed cuff tissues between the greater tuberosity and the acromium and coracaohumeral ligament, repeated motions under load conditions lend toward further tendinous changes *and* articular degenerative changes.

Osteoarthritis presents a damaging disabling condition of any joint, the shoulder joint notwithstanding. Osteoarthritic changes do not occur with the same frequency in the shoulder joint as they do in other joints. As an orthopedic pathologic entity, an osteoarthritic change presents less disability, but its role in cuff degeneration is a predominant component in shoulder lesions.

Osteoarthritis is essentially a progressive loss of cartilage, with bony and fibrous overgrowth of adjacent articular surfaces. Trauma, with resultant repetitive microfractures of the cartilage, is considered to play a predominant role in the cause of degenerative osteoarthritis. Mechanical overload causes ruptures

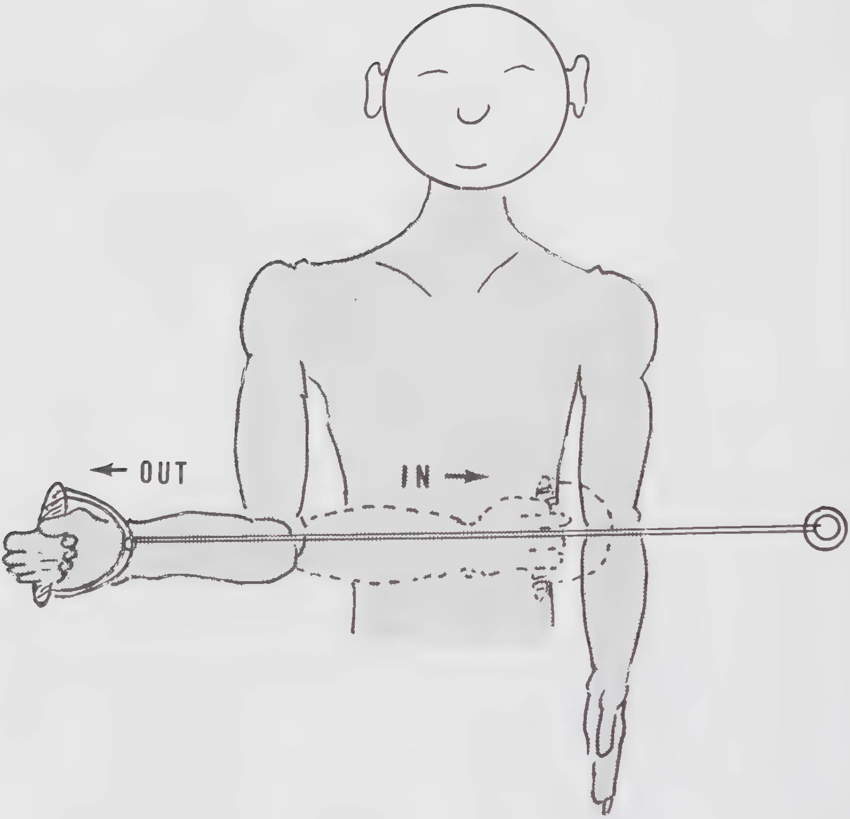


Figure 2–30. External rotator strengthening (supraspinatus muscle). With the patient sitting or standing and the affected arm flexed at the elbow 90° to the side, the hand is rotated externally as far as possible. Resistance is applied by an elastic (spring coil) securely fastened at the other end. From a full internally rotated position (*dotted line drawing*) to a full external rotation this exercise is done in increments of gradual external rotation. Strength develops depending on the increasing resistance of the elastic band, and endurance develops as a result of frequency of exercise.

in the cartilage. There are other factors that also contribute to osteoarthritic changes, including metabolic enzymatic and inflammatory changes (Huskisson and others).

A combination of etiologic factors may contribute to the eventuality of degenerative osteoarthritis in which trauma releases destructive enzymes. This enzymatic sequela favors degeneration from repetitive microtraumas.

Osteoarthritis of the acromioclavicular joint, which adversely affects the rotator cuff and enhances the painful arc entrapment, originates in earlier life—often by the second decade. It has been stated that after age 50 os-

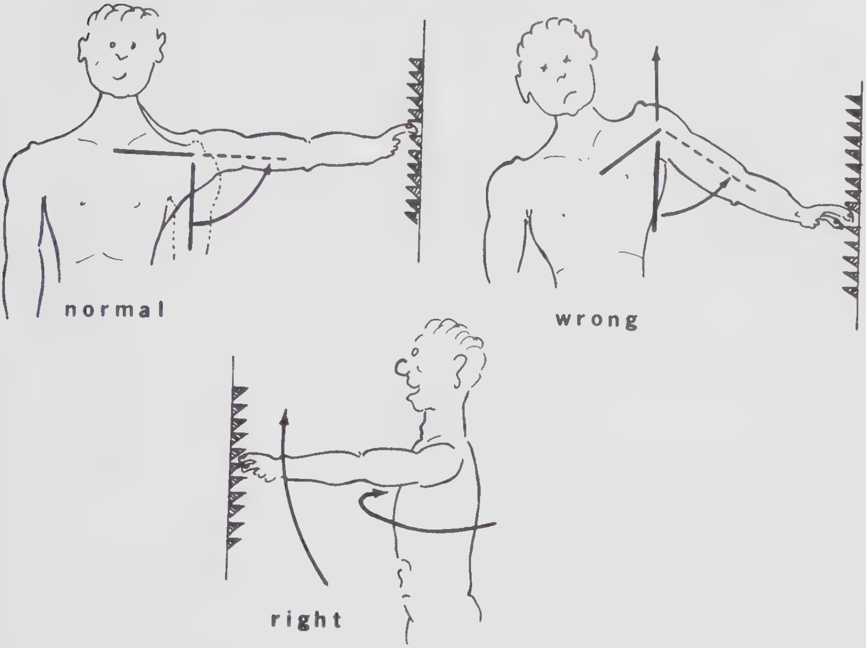


Figure 2–31. Correct and incorrect use of *wall-climbing* exercise. The wall-climbing exercise frequently is done improperly. The normal arm climbs with normal scapulohumeral rhythm. When there is a pericapsulitis, the wall climb in abduction is done with *shrugging* of the scapula and thus accomplishes nothing. The wall climb should be started facing the wall and gradually turning the body until the patient is at a right angle to the wall.

teoarthritic changes of the acromioclavicular joint are universally present (DePalma, Peterson). No relationship to the incidence of degenerative changes in the A-C joint has been proposed (Worchester and Green).

Tendons are repeatedly subjected to trauma, specifically mechanical tension, which they are structurally capable of enduring. At their site of bone insertion tendons are usually slightly thinner and fan-shaped rather than a parallel alignment of collagen fibers. Trauma resulting in damage includes paradoxic ischemia (Konn and Everth), microruptures from external trauma, vibration, and cold. Inflammation from any source leads to deposition of calcium, which adds to the mechanical and chemical trauma.

A tendon, such as the supraspinatus tendon, consists of parallel arrangement of collagen fibers which have a high resistance to elongation by tension. One tenth of the tendon consists of elastic fibers, which give it its elasticity. Fibroblasts and fibrocytes exist between bundles of collagen fibers and are considered the source of collagen nutrition, formation, and regeneration.

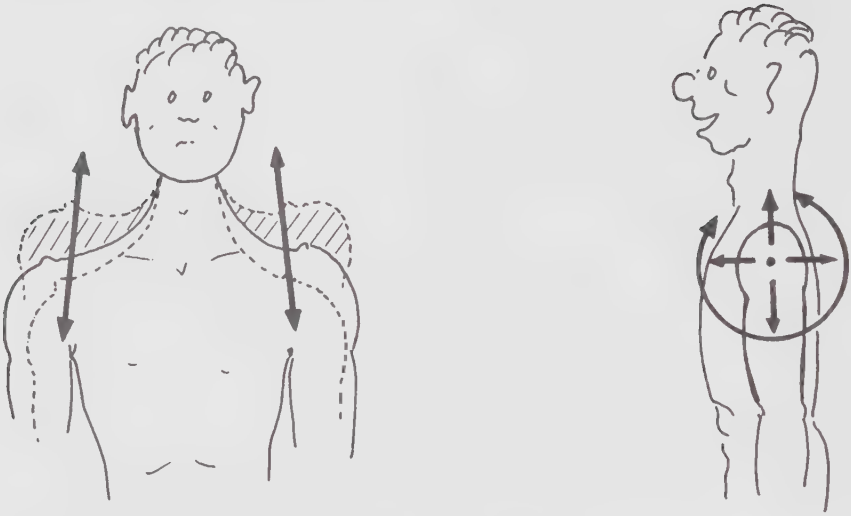


Figure 2–32. Scapular mobility exercises. Previous exercise mobilizes the glenohumeral as well as the scapulothoracic articulations. Motion here is elevation, forward and backward motion, then circumduction of the shoulder girdle. Motion here also increases sternoclavicular and acromioclavicular range of motion.

Ischemia has been proposed as the culprit of degeneration. The avascular (ischemic) zone of the supraspinatus tendon has been discussed. The blood flow to the tendon comes from the blood vessels in the bone and the adjacent muscular arterial supply. High muscular pressure and sustained muscular tension decrease the muscular blood flow to its tendon. Increased tension within the tendon also decreases its blood flow (Schatzner and Banemark).

Recent studies (Jarvholm and colleagues) have shown that at 30° of abduction the intramuscular pressure within the supraspinatus muscle exceeds the circulation pressure. This indicates that at this pressure there is impairment of the supraspinatus muscle and its tendon. There is further vascular decrease from increased intramuscular pressure at 60° and 90° abduction (Jarvholm and colleagues).

From 60° to 120° abduction (the arc distance) there is probably also further impairment of the tendon muscular blood supply as the tendon passes under the acromial process and the coracoacromial ligament.

Repetitive contractions cause tendon inflammation due to fibrin exudate. This has been demonstrated experimentally in rabbits (Rais) and confirmed in human volunteer studies (Hagberg 1985). Systemic infection has been stated to enhance degenerative changes within the tendon as a result of autoimmune reaction (Hagberg 1987).

It must be remembered that the shoulder tendons and bursae can be specifically involved in general rheumatoid disease such as rheumatoid arthritis or ankylosing spondylitis.

When discussing the supraspinatus tendon, it must be remembered that the shoulder muscles causing pain and impairment are also related. Studies of the trapezius muscles, which are predominant in supporting the scapula and which participate in scapulohumeral rhythm, reveal defects in the upper part of the trapezius described as *moth-eaten fibers* early in the life of an otherwise healthy individual (Hagberg and associates). This is considered to indicate that these fibers are performing an unusual function and are exposed to excessive traumatic stress.

An excellent evaluation of occupational shoulder and neck disorders sponsored by the Swedish Work Environmental Fund has been presented by Hagberg and merits study (Hagberg 1987b).

Pathologic progression from tendinitis to partial or complete rotator cuff tears is frequently experienced by the patient as a result of activities of daily living or occupational or athletic endeavors. The symptoms simulate those of tendinitis but merit separate consideration (see Chapter 3).

In spite of these potential detrimental effects of sustained muscular contraction and simultaneous tendon tension, there is evidence that proper exercise has a beneficial effect on a tendon.

BIBLIOGRAPHY

Bjelle, A, Hagberg, M, and Michaelsson, G: Clinical and ergonomic factors in prolonged shoulder pain among industrial workers. Scand J Work Environ Health 5, 205, 1979.

Bonica, JJ: The Management of Pain, Vol 1 and 2, ed 2. Lea & Febiger, Philadelphia, 1990.

Codman, EA: Rupture of the supraspinatus tendon: 1834–1944. J Bone Joint Surg 19A: 643, 1937.

Decker, JH: Primer on the rheumatic diseases. JAMA 190(6):118, 1964.

DePalma, AF: Degenerative Changes in the Sternoclavicular and Acromioclavicular Joints in Various Decades. Charles C Thomas, Springfield IL, 1957.

DePalma, AF: Surgical anatomy of the rotator cuff and the natural history of degenerative periarthritis. Surg Clin North Am 43:1507, 1963.

Hagberg, M, et al: Arbetsrelaterade muskulara sjukdomar: Diagnosik, behandling och prevention. Undersokningerapport 33:6, 1985.

Hagberg, M: Work load and fatigue in repetitive arm elevations. Ergonomics 24:543, 1981.

Hagberg, M: Shoulder pain-pathogenesis. In Hadler, NM: Clinical Concepts in Regional Musculoskeletal Illness. Grune & Stratton, New York, 1987a, pp 191.

Hagberg, M: Occupational shoulder and neck disorders. The Swedish Work Environmental Funds, No 40-19-17. Umea, Sweden, 1987b.

Hagberg, M and Kvarnstrom, S: Muscular endurance and electromyographic fatigue in myofascial shoulder pain. Arch Phys Med Rehabil 65:522, 1964.

Heberts, P, Kadefors, B, and Anderson, G: Shoulder pain in industry: An epidemiological study on welders. Acta Orthop Scand 52:299, 1979.

Huskisson, EC, et al: Another look at osteoarthritis. Ann Rheum Dis 38:423, 1979.

Jarvholm, U, et al: Intramuscular pressure in the supraspinatus muscle. J Orthop Res 1986.

Konn, G and Everth, HJ: Morphologie der spontanen Sehnenerreisung. Unfallheilkunde 91:255, 1967.

Kottke, FJ, Pauley, DL, and Ptak, RA: The rationale for prolonged stretching for correction of shortening of corrective tissue. Arch Phys Med Rehabil 47:345, 1966.

MacNab, I: Rotator cuff tendonitis. Ann R Coll Surg Engl 53:271, 1973.

McLaughlin, HL: Lesions of the musculotendinous cuff of the shoulder, III: Observations of the pathology, course and treatment of calcific deposits. Ann Surg 124:354, 1946.

Mosely, HF and Goldie, I: The arterial pattern of the rotator cuff of the shoulder. J Bone Joint Surg 45B:780, 1963.

Neer, CS: Cuff-tear arthropathy. J Bone Joint Surg 65A(9):1232, 1983.

Neer, CS: Impingement lesions. Clin Orthop 173:70, 1983.

Neviaser, JS: Adhesive capsulitis of the shoulder. J Bone Joint Surg 27:2, April, 1945, p. 211.

Neviaser, RJ: Treating patients with rotator cuff tears. J Musculoskeletal Med April, 1985, pp 17.

Peterson, CJ: Degeneration of the glenohumeral joint. Acta Orthop Scand 54:277, 1983.

Rais, O: Peritenomyositis (peritendinitis) crepitans acuta. Acta Chir Scand (Suppl) 268:1, 1961.

Rathburn, JB and MacNab, I: The microvascular pattern of the rotator cuff. J Bone Joint Surg 52B:540, 1970.

Schatzner, J and Banemark, PI: Intravital observations of the microvascular anatomy and microcirculation of the tendon. Acta Orthop Scand (Suppl) 126, 1965.

Simon, WH: Soft tissue disorders of the shoulder: Frozen shoulder, calcific tendonitis and bicipital tendonitis. Symposium on Surgery of the Shoulder. Orthop Clinics North Am 6(2) 521, 1975.

Watson-Jones, R: Adhesions of joints and injury. Brit Med J:1, May, 1936.

Worchester, JN and Green, DP: Osteoarthritis of the acromioclavicular joint. Clin Orthop 58:69, 1968.

CHAPTER 3

Rotator Cuff Tear: Partial and Complete

Rupture of the rotator cuff, which was originally considered to exist only as a result of severe trauma, has now become accepted as a frequent occurrence from daily activities. Today the biomechanical and pathophysiologic basis of cuff rupture is better understood. Treatment, as well as prevention, is becoming better accepted and standardized.

The mechanics of the shoulder complex has been thoroughly discussed. The causation and symptoms of supraspinatus tendinitis have also received full consideration. The progression of tendinitis to tearing of the tendon collagen fiber needs further edification, however (Fig. 3–1).

Summary of the cycle culminating in rotator cuff rupture can be derived as degenerative thinning and fissuring of the cuff in the hypovascular zone (ischemic, or avascular, critical zone) exposed to impingement or direct trauma and consequently leading to tearing of the cuff fibers. The issue as to which aspect precedes fiber disruption—degenerative changes or mechanical trauma—is conjectural. Both probably proceed simultaneously and are interdependent. Genetic factors as well as metabolic factors are present, and daily activities are added mechanical traumas. Aging factors are always imputed but not clearly delineated, inasmuch as they vary at different ages and in different activities of daily living.

Once weakened by degenerative changes, the fibers become elongated, and the mechanical fulcrum of the rotator cuff is diminished. The normal scapulohumeral rhythm is disrupted, and further trauma is imposed on the cuff from otherwise normal motion.

Five mechanisms leading to cuff rupture have been postulated (Bateman):

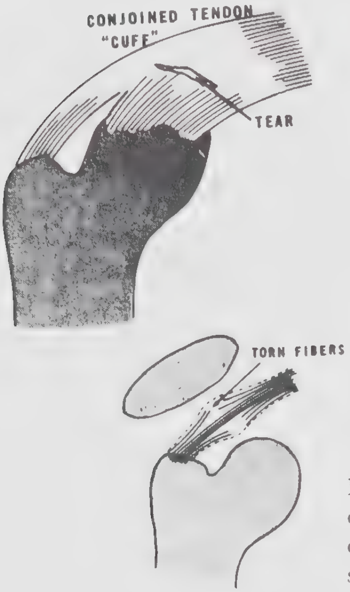


Figure 3–1. Cuff-tear. *(Top)* Site and direction of partial cuff tear. *(Bottom)* Retraction of torn cuff fibers forms a thickening of the cuff, thus resembling the thickening of tendinitis.

1. Relatively minor trauma imposed upon a degenerative cuff. This probably is the most common cause of rupture.
2. Direct fall upon the outstretched arm, transmitting the damaging force of the humeral head up against the acromial arch. Direct impingement of the cuff results.
3. Impingement of the cuff against the acromium and coracoacromial ligament from faulty abduction-external rotation overhead elevation. The major defect here is inadequate commensurate external rotation as the arm abducts so that the greater tuberosity does not pass behind but, rather, impinges upon the acromial arch.
4. Direct fall upon the arm which remains at the side, but the impact is on the anterior portion of the humeral head, causing posterior subluxation as well as direct trauma to the exposed greater tuberosity and tendon insertion.
5. Iatrogenic manipulation from attempt to forcefully free a frozen shoulder.

A rotator cuff tear can occur from a shoulder dislocation. The dislocation will receive primary concern and attention and the tear will be ignored. After reduction of the dislocation, a tear must always be considered as having occurred and must be evaluated. A fracture of the humeral head also presents this possibility.

Rotator cuff tears occur most frequently between the ages of 45 and 65 from trivial trauma, but when it occurs at a younger age the trauma is usually more severe and the tear more significant.

PHYSICAL FINDINGS

Careful history may be revealing if injury was caused by a recalled trauma and especially when it was caused by a trivial one. Pain may be elicited from specific motions of the arm during daily activities, particularly a special recreational activity, such as tennis or swimming. Pain may be nocturnal, often interfering with normal sleep.

Examination may reveal tenderness over the greater tuberosity. This can be easily identified as being immediately under the inferior edge of the acromium and lateral to the bicipital groove.

Pain can be reproduced by resisting external rotation if the tear is in the supraspinatus muscle. Pain that is elicited upon internal rotation implies a tear in the subscapularis muscle. Patients with a chronic partial tear may adbuct and/or externally rotate the arm, but weakness is determinable when compared with the contralateral side. Atrophy may be noted in the supraspinatus and infraspinatus fossae of the scapula but, when noted, indicates that the age of the tear is several weeks.

PARTIAL OR COMPLETE TEAR

The possibility of a complete tear must be initially suspected, verified, and appropriately treated. The suspicion of a partial tear rather than a complete tear must also be entertained after an acute onset of an impaired painful shoulder.

At surgery there are two main types of tear to be identified: In partial tears the undersurface of the rotator cuff is hyperemic and there is a piling up of fragmented fibers. In the complete tear there is disruption of the cuff and the inner layer of the subdeltoid synovium and the capsule (Della Porta and Evarts).

If there is no compelling evidence of a complete tear, the clinical question must arise: Is the diagnosis tendinitis with degenerative changes and elongation of the cuff, or has there been a tear in the conjoined tendon? The next question is: Is it important, at first, in diagnosis and recommended treatment, to question whether a partial tear has occurred from the incident?

The question of a partial tear is academic, inasmuch as the initial treatment will be similar to that for tendinitis. Only when symptoms and impairment persists or is greater than anticipated will the question of the existence of a partial tear be significant.

In the event of prolonged symptoms in spite of appropriate therapy the examination can reveal the extent of the tear, that is partial or complete (Fig. 3–2). Pain is not a determining factor, but active motion is. In a partial tear, albeit

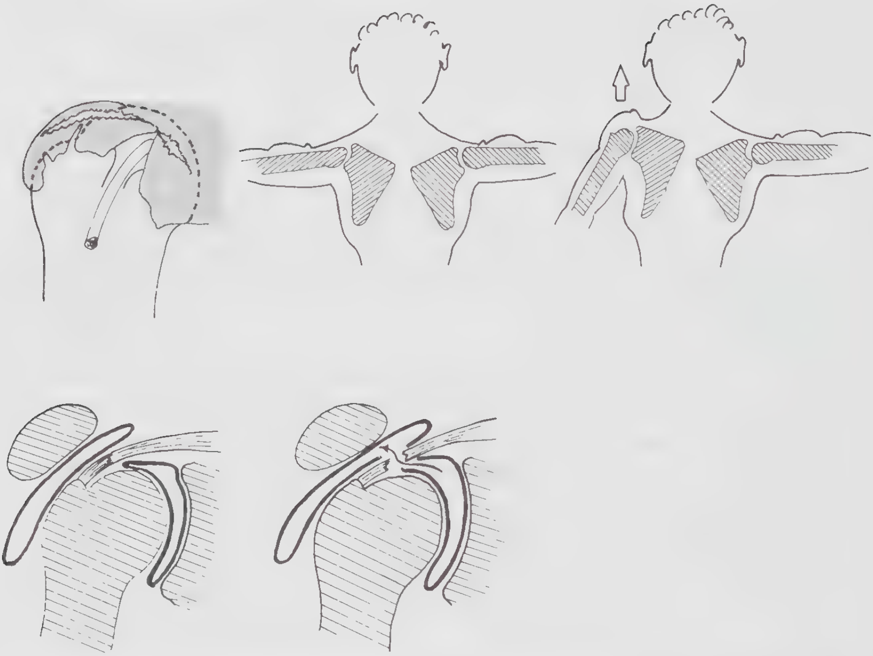


Figure 3–2. Cuff tear. The upper left diagram indicates the usual site of the tear, either partial or complete. The center posterior view of the patient abducting the arm indicates normal or even adequate scapulohumeral movement with a large but incomplete tear. The right view shows the complete tear. The lower diagrams show (*right*) the communication between the shoulder joint in a complete tear, and (*left*) the lack of communication in a partial tear or degenerated tendon.

moderate to marked, any remaining attached fibers are capable of initiating abduction and external rotation (see Fig. 3–1). Although they are weak by comparison, the fact that some abduction and external rotation are possible indicates some remaining attachment of rotator cuff fibers.

Pain may prevent any abduction or external rotation. Fear by the patient may also confuse the examiner. The very contraction of the rotator muscles may be noted, but active motion of the humerus may not be evident. Here the examiner can negate the factor of pain.

Injection of an anesthetic agent, along with reassurance and gentleness during the examination, can dispel the reluctance by the patient to actively participate. Passively abducting the arm to only 15° to 20° and having the patient hold may be accomplished by the deltoid muscle, but resistance of external rotation after passive rotation of 15° to 20° can be done only by the rotator cuff, not the deltoid. Weakness or inability to abduct the first 15° to 30° away from the side implies a total tear.

If active abduction is not elicited, the arm may then be abducted passively to 90°. At this degree of abduction the patient is then asked to hold the arm at that level. Even with a complete tear the arm can be held because at this point the deltoid is in a physiologically functional degree of shortening. The rotator cuff, however—not being involved because of total tearing—cannot seat the head of the humerus, thus the arm will slowly descend in spite of the strong deltoid action. This can be considered a *positive drop arm test* result (Fig. 3–3).

Resistance upon the abducted arm after placing it at 90° of abduction enhances the rapidity and degree of drop. It confirms that the tear is complete.

In performing the passive abduction drop arm test of rotator cuff attachment, the supraspinatus muscle has been tested as an abductor. To enhance this test a modification of the drop arm test can be initiated. This enhanced test evaluates the efficacy of external rotation, which is also a supraspinatus function.

Placing the shoulder actively or passively in 90° of abduction (horizontal), (Fig. 3–4) as in the drop arm test, the position allows the deltoid to act. Now the arm is flexed 90° at the elbow, placing the hand and forearm in a horizontal position. The muscles that now must act to maintain this externally rotated position of the arm are the rotator cuff muscles. Any pressure downward upon the hand-wrist will be resisted if the rotator cuff is effective. If the cuff has been torn and therefore disconnected from the greater tuberosity, the weight of the forearm will cause it to descend gradually (internally rotate). Pressure downward upon the forearm will precipitate the downward rotation.

In the presence of a complete tear the forearm cannot be held in the horizontal position, even briefly, with any downward pressure from gravity or from the examiner. There is a positive forearm drop test result in a complete tear. In a partial tear there is weakness elicited by downward pressure as compared with the contralateral (normal) arm. This, too, can be considered a positive forearm drop test result, even though the forearm does not drop but merely descends.

Confirmatory Test of Cuff Tear

Once there is clinical suspicion and substantiation of cuff tear, confirmatory tests are available to verify the presence and extent of the tear.

Arthrography has been shown to be effective (Fig. 3–5). Normally, when the glenohumeral joint is injected with a contrast medium, the entire content of the injected dye is retained within the glenohumeral capsule. The bursae of the shoulder complex are not invaded by the injected dye. If there is a tear in the rotator cuff, the dye injected into the glenohumeral capsule passes through this tear into the subdeltoid bursa. Flow into the subdeltoid bursa indicates a direct communication possible only by a tear in the conjoined tendon. The degree of tear is thus measurable.

More recently magnetic resonance imaging (MRI) testing has been shown to be very effective in diagnosing a rotator cuff tear without invasion. The MRI indicates the presence of the tear but not the degree of tearing.

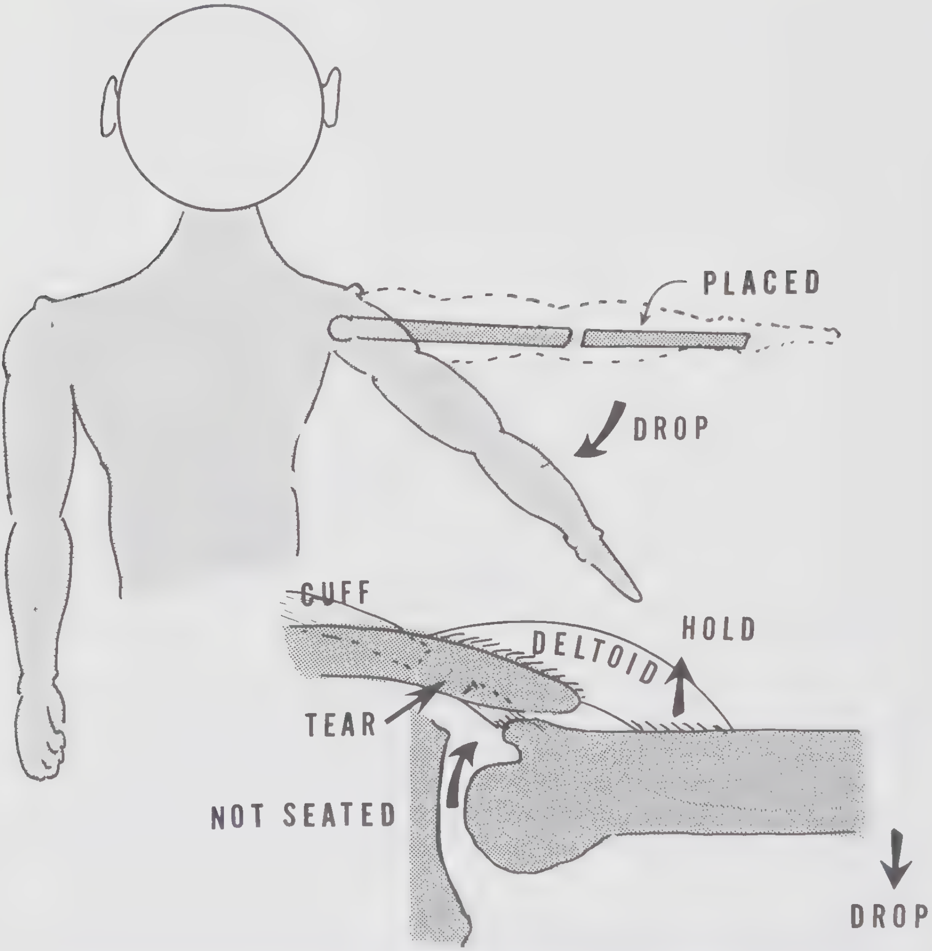


Figure 3–3. Drop arm test for rotator cuff tear. In a drop arm test with a positive result the deltoid muscle holds the arm in abduction but, because of the absence of rotator cuff attachment, the humeral head is not held firmly seated in the glenoid fossa and thus rotates upward. The arm then drops from the abducted position. Merely having the patient sustain the arm, which the deltoid will do, shows that the arm remains abducted briefly and slowly lowers. With superincumbent downward pressure the arm literally drops.

TREATMENT OF THE ROTATOR CUFF TEAR

Treatment of the acute complex rotator cuff tear is universally accepted to be surgical intervention in the younger physically active group. The only controversy is the determination of *younger* and *physically active*. Many cases have been successfully repaired surgically in people 60 to 70 years of age. The efficacy

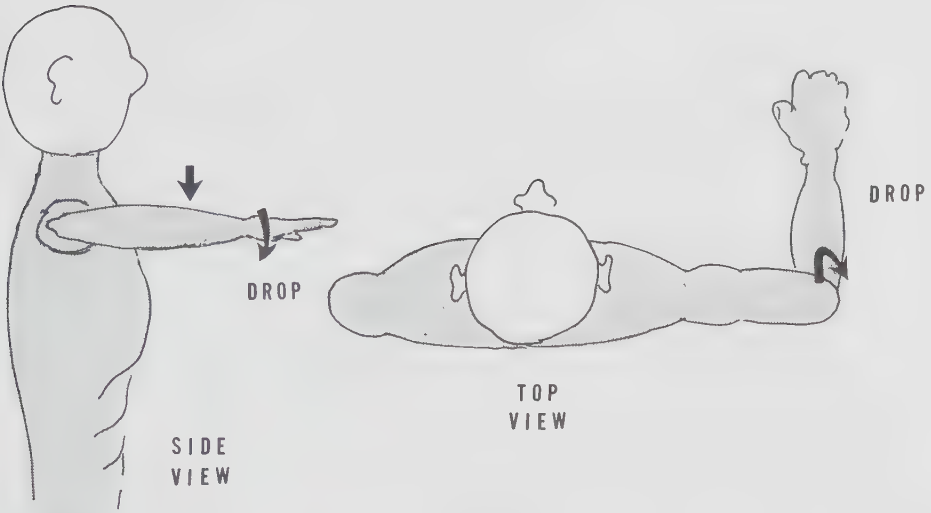

DROP
DROP
TOP VIEW
SIDE VIEW

Figure 3–4. Forearm drop test for rotator cuff tear. A modification of the drop arm test can be termed the *forearm drop test*. This is essentially a test of external rotation of the rotator cuff. If the cuff is torn, as the arm is held in abduction, flexed 90° at the elbow, the forearm will fall (drop) into internal rotation or drop down from downward pressure upon the forearm.

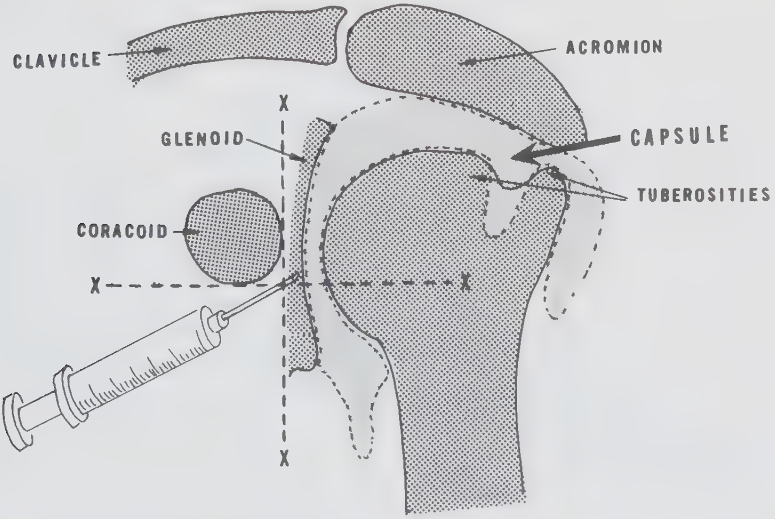

CLAVICLE
ACROMION
X
GLENOID
CAPSULE
TUBEROSITIES
CORACOID
X
X
X

Figure 3–5. Injection technique for intra-articular arthrogram and brisement treatment. The injection site is a point just inferior and lateral to the coracoid process in the vector lines (X-X).

of repair is dependent upon the ability of the cuff tissue to respond to resection and suturing.

The technique also varies as to the arthroscopic approach versus the open arthrotomy. The expertise and experience of the consulting orthopedic surgeon are the definitive factors here.

Proponents of the conservative nonsurgical approach to the chronic partial rotator cuff tear claim that in time the inflammatory reaction of the cuff subsides and the integrity of the remaining cuff fibers function better than if there has been surgical intervention. In time the thickness of the torn and self-repaired fibers subside and there is decreasing entrapment.

Routine excision of the intra-articular portion of the biceps tendon has the advantage that there will be no ultimate bicipital adhesion syndrome following acromionectomy, and the enlarged biceps tendon does not become a source of entrapment.

Proponents of surgical intervention claim that the repair enhances the mechanical advantage of the cuff and that the debris can be excised, presenting less mechanical obstruction to glenohumeral action. By surgical intervention of the rotator cuff the possibility of adhesive capsulitis and adhesive bursitis (*frozen shoulder)* is allegedly enhanced (Simmonds).

Proponents of surgical intervention also state that the mechanical obstructive tissues of degenerative arthritis of the acromium and of the acromioclavicular joint can be altered. Acromionectomy is favored by many, but rejected by an equal number (Neer). The degree of acromionectomy plays a major role in its advantage versus disadvantage (Neer) (Fig. 3–6). A *radical* acromionecto-

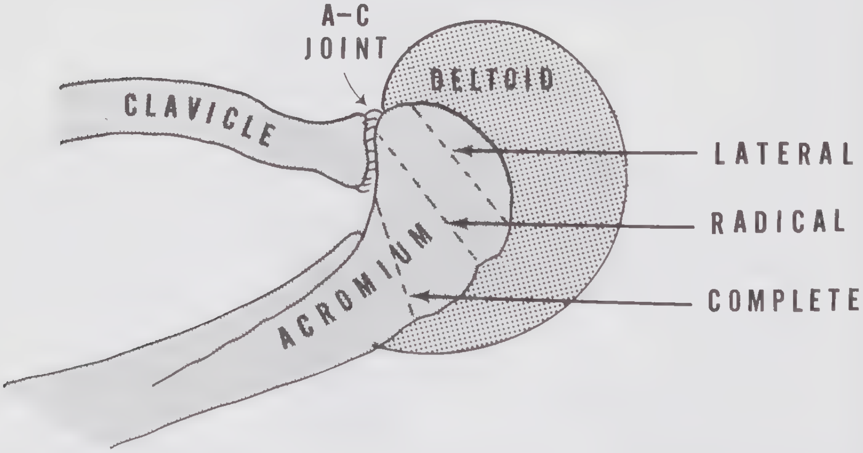


Figure 3–6. Sites of acromionectomy. The surgical decision of degree of acromionectomy has been divided into lateral, radical, and complete. The latter removes the acromioclavicular (A-C) joint completely.

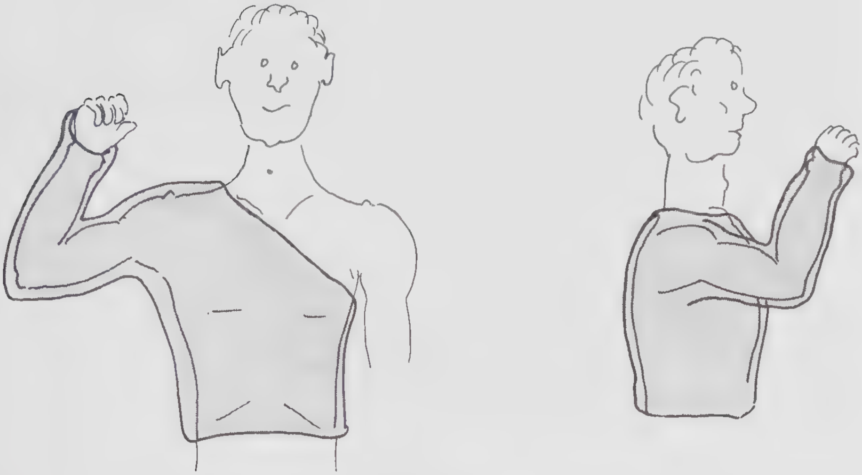


Figure 3–7. Proper spica cast for the torn cuff treatment. The body spica must hold the arm (humerus) abducted, flexed forward, and externally rotated. This is the position of least tension upon the rotator cuff and therefore that of maximum opposition of the torn ends of the cuff.

my decreases the amount of attachment of the deltoid muscle and renders it ineffectual. Too much removal of the acromium also exposes the rotator cuff, rendering it superficial and covered only by skin. If the cuff has been surgically repaired, it is exposed to direct trauma by this exposure.

The patient must be fully informed of the pros and cons of the surgical intervention as compared with those of conservative management. Postoperative management is very important to the success of the surgical procedure. The length of immobilization and the manner of splinting will differ according to the extent of surgery.

Immobilization in a body cast (Fig. 3–7) is advocated by some, but it can be seen that prolonged immobilization in this type of splint threatens joint contracture of the shoulder and the elbow and may lead to excessive atrophy of all the upper extremity muscles.

As dictated by the surgeon (Codman), passive pendular exercises can be initiated, followed by the routine therapy advocated in supraspinatus tendinitis. The repaired tendon is considered strong at 6 weeks.

BIBLIOGRAPHY

Bateman, JE: The diagnosis and treatment of ruptures of the rotator cuff. Surg Clin North Am 43:1523, 1963.

Codman, EA: The Shoulder. Thomas Todd, Boston, 1934.

Della Porta, RJ, and Evarts, CM: Rotator cuff tears. Contemp Orthop 4(3):327, 1982.

Neer, CS, and Marberry, TA: On the disadvantages of radical acromionectomy. J Bone Joint Surg 63-A(3):416, 1981.

Neviaser, RJ: Treating patients with rotator cuff tears. J Musculoskeletal Med, April, 1985, p 17.

Rosenberg, PS, and Clarke, RP: Chronic rotator cuff tears. Orthop Review (5):33, 1986.

Simmonds, FA: Shoulder pain with particular reference to the "frozen" shoulder. J Bone Joint Surg 31B: 3, Aug 1949, p 426.

CHAPTER 4

The Frozen Shoulder: Adhesive Capsulitis, Adhesive Bursitis

The feared sequelae of shoulder tendinitis, bursitis, partial tear, or even reflex sympathetic dystrophy is the *frozen shoulder.* A shoulder that is initially painful but which gradually becomes more restricted in motion in all directions is considered frozen.

The condition widely claimed to be a frozen shoulder still remains an enigma as to exact origin, tissue involvement, causation, mechanism, and the ideal forms of prevention and treatment.

The numerous concepts are evident when the diagnostic labels applied to this condition are reviewed: adhesive capsulitis, adhesive bursitis, periarthritis, pericapsulitis, obliterative bursitis, stiff shoulder, scapulohumeral periarthritis, Duplay's disease, and many others.

It appears that many tissues, mainly synovial, are involved in the ultimate frozen shoulder (Fig. 4–1). These include any or all of the following:

1. The synovium of the subdeltoid bursa
2. The synovial lining of the glenohumeral capsule
3. The tenosynovium of the conjoined tendon
4. The synovial lining of the biceps tendon
5. Subscapularis muscle and the subscapularis bursa

The synovial layers of the subdeltoid bursa are contiguous with the outer synovial layer of the conjoined tendon. The inner synovial layers of the conjoined tendon are contiguous with the glenohumeral capsule synovium and with

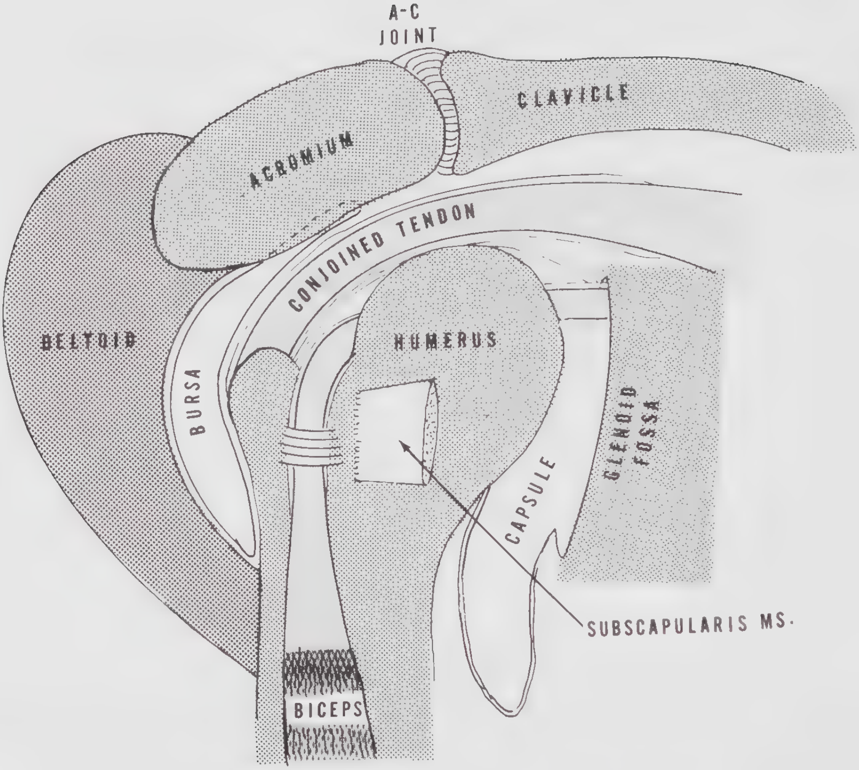


Figure 4–1. Tissue sites within the glenohumeral joint space for formation of *adhesions.* All the tissues have been listed. The synovial layers are depicted as fine lines layering all the enclosed tissues.

the tenosynovium of the biceps tendon.

All these tissues are contained within a small compartment between the head of the humerus, the acromioclavicular joint, and the coracoacromial ligament. Each constantly moves upon the other in a gliding manner during any and all shoulder motions. These tissues are apparently well lubricated and dependent on frequent continual motion.

Pain is apparent early in this syndrome and enhances the ultimate tissue changes. This is schematically summarized in Figure 4–2.

The term *periarthritis* was first described by Duplay in 1906 when he described a shoulder condition now recognized as the frozen shoulder. It was Codman who first made an extensive study of this clinical entity. He considered this condition as due to adhesions of the subacromial bursa and later related this condition to tendinitis of the rotator cuff tendon.

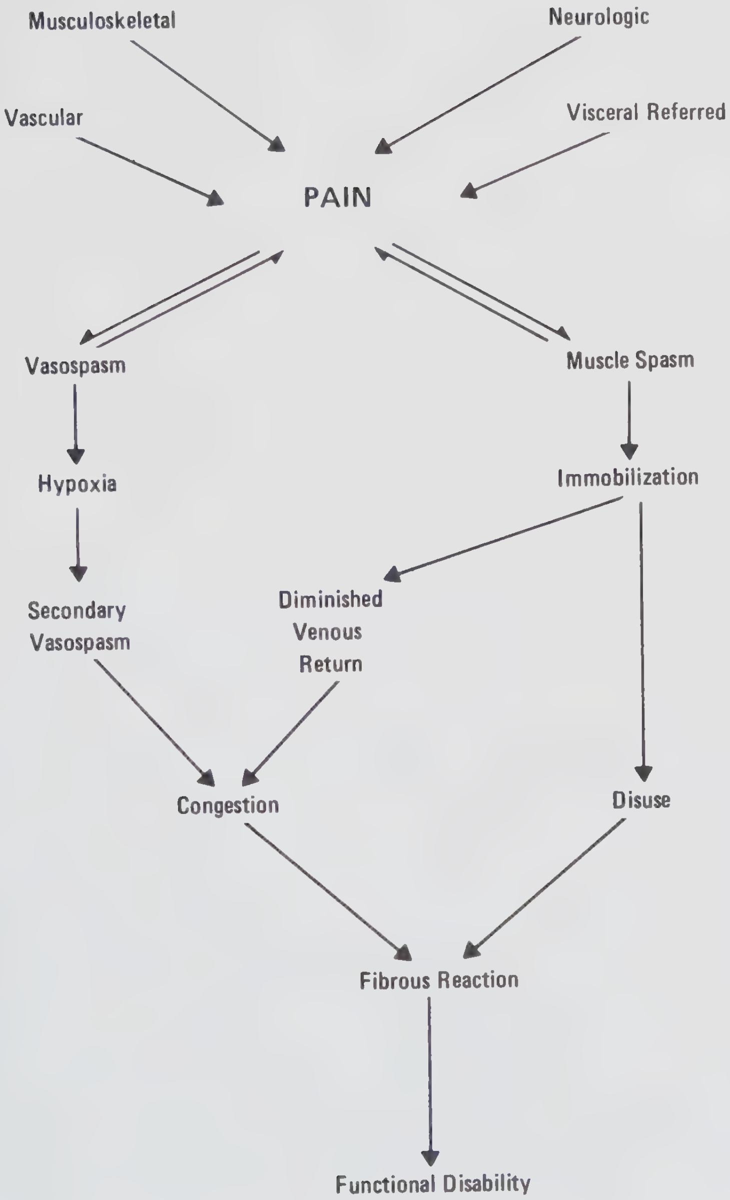

Musculoskeletal
Neurologic
Vascular
Visceral Referred
PAIN
Vasospasm
Muscle Spasm
Hypoxia
Immobilization
Secondary Vasospasm
Diminished Venous Return
Congestion
Disuse
Fibrous Reaction
Functional Disability

Figure 4–2. Schematic stages toward functional disability in the painful shoulder.

Horowitz first did intensive studies of afflicted shoulders. He described chronic thickening of the bursa with formation of villi and fraying and fibrillation of the cuff. He found constrictions of the biceps tendon that otherwise was essentially normal, albeit abnormally attached to various portions of the shoulder complex. He also found bony changes in the undersurface of the acromium and the bicipital groove. It can be assumed from his conclusions that he attributed the restriction of glenohumeral motion, the frozen shoulder, to adhesive biceps tendinitis. Lippman and DePalma stated, in their papers, that removal of the intra-articular portion of the biceps tendon immediately freed shoulder motion.

Lippman actually attributed shoulder pericapsulitis to biceps tendinitis and claimed that relieving the inflammation of the biceps tendon would obviate the ultimate adhesive restriction of all the periarticular tissue.

DePalma and colleagues essentially agreed on this sequence of conditions. Their studies revealed gradual marginal bone proliferation about the glenoid labrum which, they felt, caused inflammation of the biceps tendon (the predominant lesion according to DePalma) and the capsular ligaments. These changes gradually led to degenerative changes of the fibrotendinous cuff, with thickening and adhesion from increased vascularity and fibrosis.

McLaughlin stressed the importance of the subscapularis muscle in the causation of the frozen shoulder. Although the supraspinatus tendon is considered the primary site of inflammatory changes, the adjacent synovium undergoes hypertrophy and increased vascularity (MacNab), and the inferior fold of the capsule gradually involves the subscapularis muscle with resultant contracture. This muscle, an internal rotator, has been impuned in the causation of the hemiplegic shoulder synergy syndrome, in which the shoulder of the completed stroke essentially freezes in an adducted internally rotated position. This is completely discussed in the chapter on the hemiplegic shoulder.

Neviaser (1945) found thickening and adhesions of the glenohumeral capsule, especially at its inferior aspect. He differed with Horowitz in that he found very few significant adhesions of the biceps tendon. He demonstrated that severing the inferior capsule and the adhesions relieved the restricted glenohumeral motion. His microscopic studies revealed degeneration of the capsular collagen fibers with increased vascularity, mononuclear cell infiltration, and fibrous adhesions. He essentially attributed the frozen shoulder to adhesive capsulitis and advocated releasing these adhesions by manipulation.

The sequence of events appeared to be gradual degenerative changes from repeated (micro) trauma, resulting in loss of the elastic aspect of connective tissue. Consequently there is an inflammatory reaction, causing granulations (Fig. 4–3) to invade the damaged area with fibroblasts. Adhesions are the result (Turek).

In essence the repeated traumas of entrapment of the rotator cuff between the greater tuberosity and the overhanging acromium and coracoacromial ligament, enhanced by gradual inefficiency of the rotator cuff muscles and the mechanical assistance of the biceps tendon, result in degeneration of the bursa and

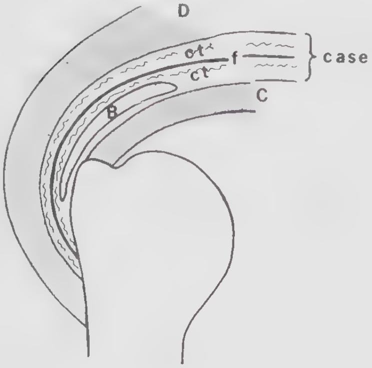


Figure 4–3. Periarticular case of the suprahumeral joint. The lining and contained tissues of the suprahumeral joint are graphically depicted as a *case*. The superior layer is the undersurface of the deltoid, and the floor is the outer surface of the rotator cuff. Interposed is loose connective tissue (ct) and a strong subdeltoid fascia (f). The fascia is richly endowed with blood vessels and sympathetic nerve endings. Proximally the fascia contains the coracoacromial ligament (not shown) and ends distally in the periosteum of the humerus at the level of the surgical neck.

the capsule. That there may be a systemic metabolic vascular component to adhesive pericapsulitis is the finding of Bridgman, who discovered a 10.8 percent incidence of frozen shoulder syndrome in a series of 800 unselected diabetic patients. It is well documented that many diabetic patients who suffer a stroke also undergo subsequently a frozen shoulder and a reflex sympathetic dystrophy condition.

Any mechanical entrapment initiates irritation causing an invasion of granulation tissue, edema, and fibroblastic activity. Repair of these tissues is delayed or impaired by frequent irritation, especially if there is a preceding degenerative change already imposed by age.

The muscles attached to the involved tendons also undergo changes of degeneration, including contraction and elongation, gradually increasing the possibility of further mechanical breakdown of the scapulohumeral rhythm.

Failure to achieve daily full range of motion accepts the failure of the capsule, being fully elongated, and that of the biceps tendon, being repeatedly moved within its fibrous tunnel (under the transhumeral ligament over the bicipital groove). As the biceps tendon becomes less mobile within its canal, it undergoes further degenerative changes and ultimately adhesion.

With gradual failure of proper abduction, external rotation implemented by the supraspinatus and deltoid muscles, and the mechanical assistance of the biceps tendon, further mechanical impingement of the greater tuberosity and the acromium and the coracoacromial ligament occurs.

The resultant bony contact results in osteoarthritic spurring of the distal process of the acromium—a cause of further mechanical trauma. Repeated mechanical irritation also thickens the anterior edge of the coracoacromial ligament, causing further mechanical trauma to the abducting externally rotating humerus.

It is apparent that many factors play a role in ultimate tendon inflammation and degeneration and in the sequence of a frozen shoulder. These roles must be examined in order to clarify the meaning of a clinical examination and to formulate a preventive program and a therapeutic approach when there has been painful limitation of the shoulder.

The tissue site of pain in this condition has not been clearly determined. There are those (Lippman and DePalma) who attribute all pain to the bicipital tendon. The relief of pain often achieved by sympathetic nerve block seems to imply that the pain is neurovascular in origin, but that, again, is conjecture. The contracture of the synovium of the subdeltoid bursa and/or the capsule has also been imputed. The exact basis of pain remains for further research to determine.

CLINICAL PICTURE: PHYSICAL FINDINGS

The entity of a frozen shoulder has rarely been reported before the age of 40, unless an acute trauma has been followed by long periods of inactivity and anxiety or tension. The usual onset occurs from 50 to 70 years of age. The sex variance differs, with women reported more frequently than men. Several attempts have been made to imply a psychologic predisposition: the so-called periarthritic personality. This, however, has not been confirmed. The syndrome is noted more frequently in sedentary workers than in laborers.

The syndrome develops insidiously, beginning with pain and tenderness usually over the deltoid insertional area in the upper outer humerus. Motion aggravates the pain, and gradually a limitation of both active and passive motion develops. What at first hampers daily activities gradually interferes with sleep.

Tenderness over the greater tuberosity has been found to exist in supraspinatus tendinitis. It may also be present in the shoulder beginning a freezing sequence. Tenderness is usually noted on palpation of the biceps tendon.

Limited active abduction is noted. The patient demonstrates the shrugging mechanism, discussed in Chapter 2 as noted early in tendinitis and in partial and complete rotator cuff tear. Considered to be caused, at first, by pain and entrapment during the painful arc, this limited motion persists and is even augmented when soft tissue adhesion begins.

In the early frozen shoulder the last 10° to 15° of motion are initially lost. The subtle signs of limitation (Fig. 4–4) are noted early and should always be recognized to ensure prompt attention and intervention. Pain as the restricting factor can be eliminated by intra-articular or suprahumeral anesthetic injection or a suprascapular nerve block. Once the pain is eliminated or diminished, the tissue limitation is now apparent.

The limited active range of motion is also evident in passive range testing. Active abduction should be tested with the arm internally and externally rotated. External rotation should also be tested with the arm dependent at the side and in any degree of upper arm abduction that can be attained.

Strength of all the active motions should also be tested, as well as range of

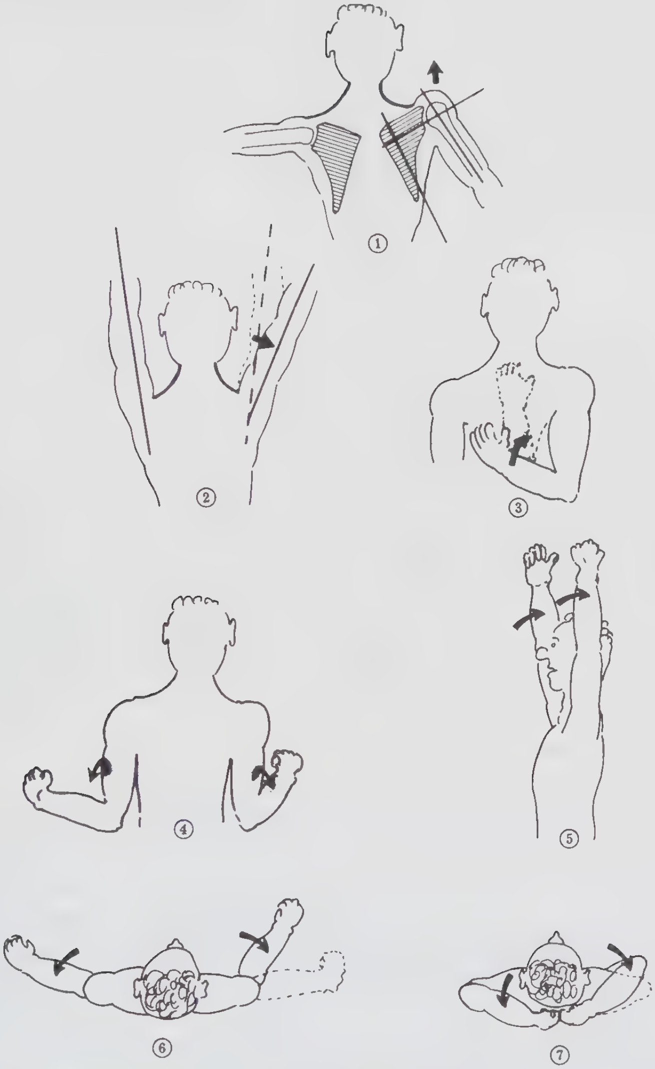


Figure 4–4. Subtle signs of shoulder limitation: (1) shrugging with excessive scapular rotation and limited glenohumeral abduction; (2) limited right arm overhead elevation (arm away from head and ear); (3) limited posterior flexion and internal rotation (hand fails to reach normal interscapular distance of reach); (4) limited external rotation of right arm, done with flexed elbow; (5) overhead elevation of right arm limited in posterior direction as compared with normal, viewed from side; (6) external rotation as viewed from above; (7) with hands behind head, failure of right arm to fully extend posteriorly.

motion. This includes the biceps as well as the scapular muscles. Atrophy may also be noted, especially in the supraspinatus and infraspinatus regions of the scapula. The presence of atrophy implies that the condition has existed for some time and that it is not necessarily an acute problem.

Compared with acute tendinitis, in the frozen shoulder there is limitation in most active and passive motions, not merely in abduction or external rotation. Pain is usually initiated—certainly aggravated—by exceeding the extremes of the gained range during the examination.

RADIOLOGIC SIGNS

X-ray evaluation may reveal no specific diagnostic findings other than some osteopenia or cystic changes in the lateral aspect of the humerus. Degenerative bony changes on routine pictures may reveal an inciting causative factor in tendinitis but not the incriminating tendons, capsule, or bursa.

An arthrogram depicts the presence and degree of adhesive capsulitis. A normal glenohumeral joint will accept 30 ml of fluid or dye, whereas in adhesive capsulitis there is some restriction of the capsule and the amount accepted decreases. The injected dye reveals this decrease in the joint space, especially the limitation of the inferior space (Fig. 4–5). It must be stated that performing an arthrogram presents technical difficulties as the entrance into the limited capsule is difficult. An MRI may rule out other abnormalities but is not specifically diagnostic of adhesive capsulitis with regard to the exact tissues involved or the severity of involvement.

TREATMENT OF THE FROZEN SHOULDER

Treatment for the frozen shoulder varies so much with experienced clinicians that currently there is no standardized treatment. This discrepancy resides in the fact that not only the degree of *freezing* determines which treatment regimen is the most appropriate, but also what pathologic process is causing the adhesions.

From the previous discussion it remains abundantly clear that there remains a great deal of controversy as to which tissues in the shoulder complex are involved and why adhesions occur. There must be some clarification, therefore, to justify specific treatment.

Benign neglect of the frozen shoulder has been advocated with the statement that "regardless of any treatment, ultimate recovery will occur within 2 years." This adage is questioned by many and thus it must be refuted. Started early and with intensity—*before* there is significant adhesion—active physical therapy is more applicable and is advocated by most clinicians who are studying and treating this condition.

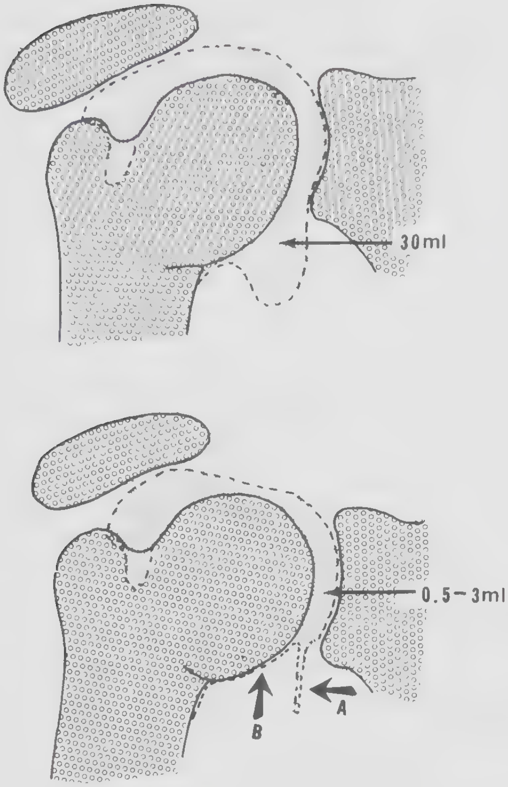


Figure 4–5. Adhesive capsulitis. The normal capsule permits injection of at least 30 ml of air. In adhesive capsulitis, the capsule adheres to itself (A) and to the humeral head (B). This decreases capacity to 0.5 to 3 ml and markedly limits range of motion.

Conditions Relating to the Frozen Shoulder

There are many conditions relating to frozen shoulder, including post myocardial infarct, post prolonged immobilization, post fracture/dislocation, and merely prolonged disuse. The reflex sympathetic dystrophy (shoulder-hand-finger syndrome) is another classic example of the frozen shoulder with complications involving the hand, wrist, and fingers. Whether the tissue inflammation mechanism is identical in all these conditions is conjectural, but undoubtedly many—perhaps most—of the tissues changes are similar. The treatment for all, therefore, should be similar.

There is even a question as to whether all frozen shoulders begin with pain and lead to restriction or begin with restricted movement that eventually becomes painful. Adhesive capsulitis, for want of a better diagnostic label acceptable to all clinicians, is a subgroup of the painful shoulder. It may present as a primary condition, or it may be secondary to other shoulder problems.

Painful Shoulder Problems

1. Supraspinatus tendinitis
2. Subdeltoid bursitis
3. Degenerative A-C arthritis
4. Adhesive capsulitis
    a. post myocardial infarct
    b. post stroke
    c. post casting: prolonged immobilization
5. Referred pain—this overlaps with 4, above.

The usual stages of evolution are pain, gradual restriction of motion, and ultimately marked limitation without pain. The last stage is a stiff, useless but painless shoulder which hurts only when forcefully moved to ensure or to determine limitation.

The major therapeutic controversy appears to be when there is significant adhesion: whether prolonged physical therapy, open reduction, brisement, or manipulation is the best treatment of this *completely* frozen shoulder (Murnaghan).

It must be accepted that with any cause of pain in the shoulder some restriction of motion occurs. In supraspinatus tendinitis with a painful arc, there is pain on abduction, on overhead elevation, and occasionally on external rotation. This occurs also in calcific tendinitis.

In biceps tendinitis the pain elicited is noted in the anterior shoulder area with simultaneous elbow flexion and supination. In the condition of acromioclavicular (A-C) arthritis or injury there is also pain on scapular elevation and circumduction of the entire shoulder.

In primary capsulitis any and all motion may cause pain. After fracture or dislocation there may be limited motion in all directions. Any specific direction of motion causing pain leads to ascertaining which specific tissue is involved. What is important, however, is that regardless of the initial lesion there is pain and limited movement. There is, therefore, a resultant total tissue reaction from the primary lesion that may remain as a potential capsulitis: a frozen shoulder. After the abating of the initial pathologic process, the remaining capsular reaction becomes the primary lesion and the cause of pain and disability. This secondary limited motion initiated by pain, regardless of initial lesions, is the basis for early initiation of the Codman passive pendular exercises advocated in Chapter 2 as mandatory to regaining and to maintaining range of motion.

During an acute episode, passive then active range of motion must be initiated, done either by the patient or, when it is too painful or difficult, with the assistance of a therapist. Pain relief is achieved by local ice (Clarke and associates), then heat, ultrasound application, transcutaneous electrical nerve stimulation (TENS) application, oral NSAIDs, and even oral analgesic medication. Mobilization and gentle manipulation (Maitland) may also be advisable in the early stage

(Figs. 4–6, 4–7). Rhythmic stabilization exercises have proven to be of value (Fig. 4–8). These are assisted and resisted exercises initiated by the patient with the help of a therapist. Each range is attempted by the patient at the instruction of the therapist (Fig. 4–9), who applies traction and resistance to the motion. Muscular contraction followed by relaxation of that specific motion is initiated. There is also reciprocal relaxation of the antagonist when there is contraction of the agonist. Because the patient actively initiates the motion, he or she has *control* and will avoid anxiety and resistance in moving the painful arm. By gradually increasing the range at each contraction, increased flexibility results.

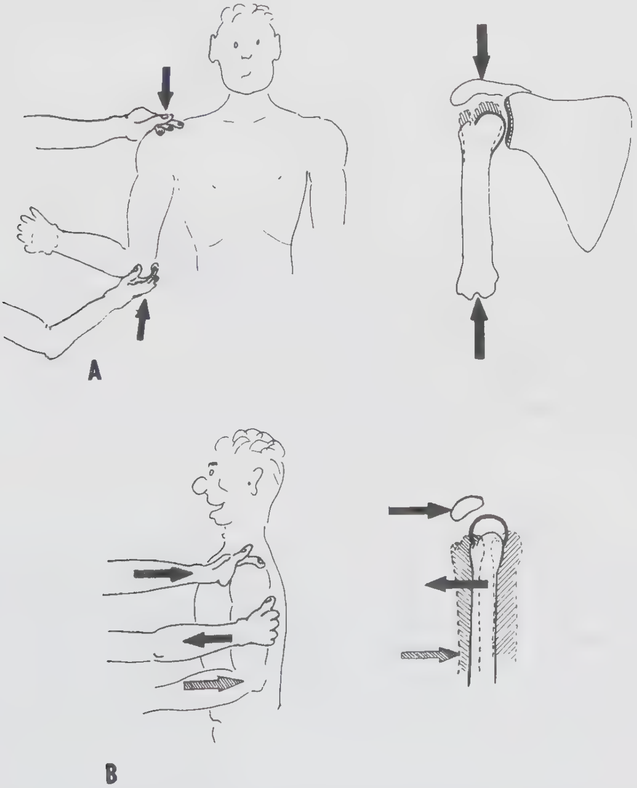


Figure 4–6. Manipulation treatment of *involuntary* motion of the glenohumeral joint. *(A)* Elevation of the head of the humerus against the glenoid fossa. Pressure along the shaft of the humerus, with the other hand preventing elevation of the scapula, causes the humerus to elevate, thus stretching the superior capsule. *(B)* Anterior and posterior motion of the head of the humerus against the glenoid. Three points of contact must be applied. One hand mobilizes the humerus while the other hand *fixes* the scapula. The elbow or forearm is fixed by the therapist's body or elbow.

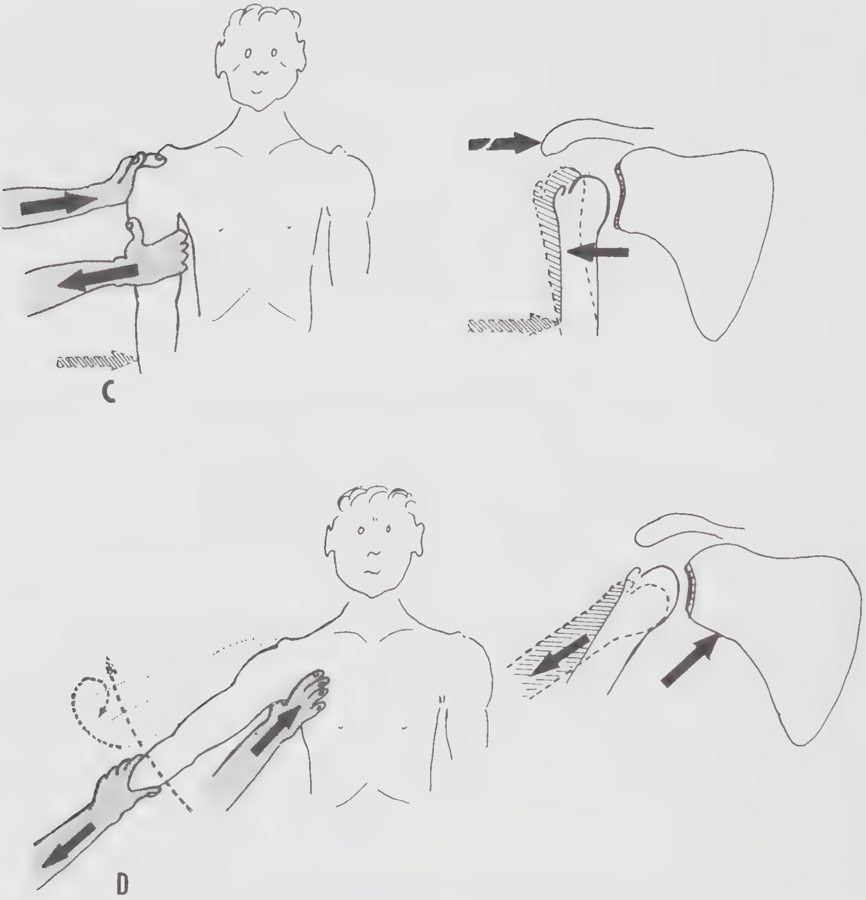


Figure 4–6 *(continued).* *(C)* Lateral motion of the head of the humerus away (in separation) from the glenoid. One hand of the therapist pulls at a right angle to the shaft of the humerus while the scapula and the elbow are *fixed.* *(D)* Traction to separate the head of the humerus from the glenoid while abducting and gradually externally rotating the arm. Counterresistance (fixation) is applied against the axillary border of the scapula.

Intra-articular injections of an analgesic agent with or without steroids are of value to begin regaining range of motion.

All aspects of treatment in the early stage of frozen shoulder, aimed to maintain or to regain full range of motion, have been discussed in the previous chapter on shoulder tendonitis. Once the shoulder is completely adherent, treatments present some controversy and differing prognoses.

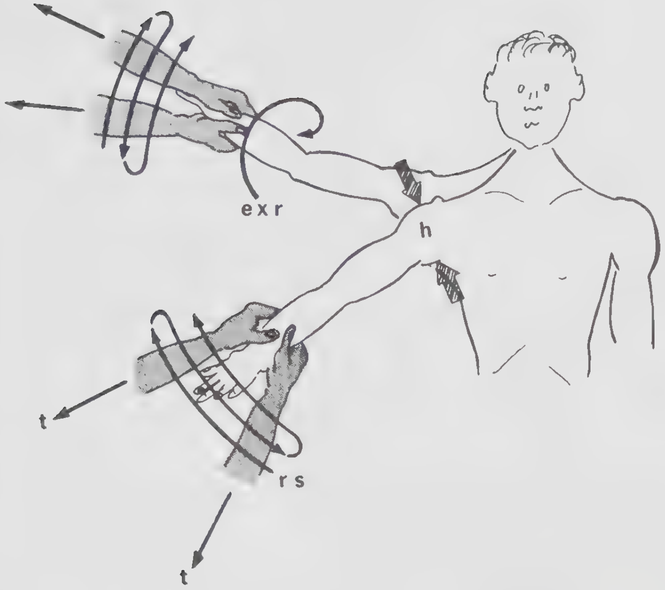


Figure 4–7. Rhythmic stabilization manipulation of the glenohumeral joint. While the patient applies isometric contraction of the glenohumeral joint to *prevent motion*, the therapist attempts to move the arm in abduction external rotation, then reverses to adduction internal rotation. Traction (t) is constantly applied. Motion by the therapist is smooth and gradually increasing then decreasing in force and amplitude, with the patient resisting it with equal force. After one cycle at a specific range of abduction, the arm is passively abducted and externally rotated to a further point, and the cycle is repeated.

Suggested Treatments

Simmonds reviewed 21 cases of frozen shoulder and found that only 6 had recovered full range of motion after 3 years. On the other hand, Reeves reported that 41 cases that were treated merely with analgesics recovered within 30 months. Grey reviewed 25 cases also treated merely with analgesics and found that 24 had painless full range of motion after 2 years.

To refute the idea that most patients with frozen shoulder eventually recover, Reeves reported that in a 5 to 10 year follow-up, over 50 percent of patients continued to have restricted motion. Clark and colleagues reported 42 percent of patients still having restricted motion after 6 years follow-up. Binder and associates noted 40 percent of patients failing to regain minimum range of motion. These controversial figures indicate the need for aggressive treatment of the frozen shoulder.

In a recent review (Ozaki and colleagues) of 17 patients treated surgically

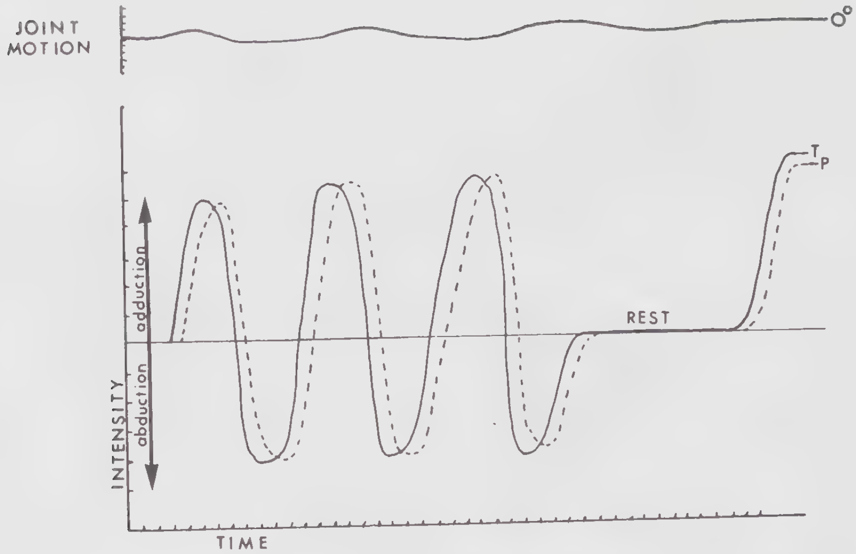


Figure 4–8. Schematic graph of rhythmic stabilization. The solid line (T) indicates the increment of force applied to the shoulder by the therapist in alternating abduction then adduction, immediately followed by opposing force from patient (P) to prevent joint motion (0°).

and followed for 6.8 years, it was found that only one had pain after overuse and one had slightly limited motion. These authors revealed that all patients treated surgically retained an opening in the capsule but no instability. Their revelation was that upon surgery they found no adherence of synovial tissue to the humeral head but, rather, adherence of the synovium to itself. This is consistent with the original concept of Neviaser.

Using constant traction combined with TENS, Rizk and colleagues found that TENS was more effective than heat with traction, but recoveries were only partial. These reviews seem to indicate that a frozen shoulder can recover within 2 to 3 years with nonmanipulative treatment. These conclusions, however, are not universally held.

The condition that exists within the glenohumeral joint determines whether to consider gradual stretching, repeated stretching, manipulation, or ultimately surgical intervention. Most of the pathologic process has been discussed.

At surgery, different findings have been claimed. Neviaser found a thickened band under the inferior capsule which he could not identify. It contracted when the arm was abducted. He found the entire capsule adherent to the head of the humerus, which he could slowly separate. He found that the inferior fold

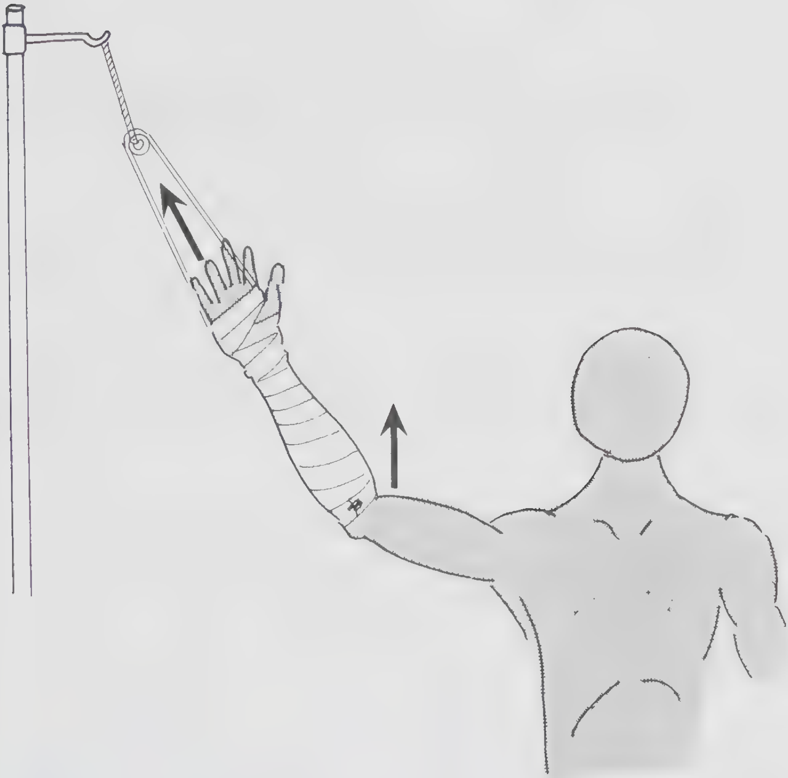


Figure 4–9. Antigravity treatment of the edematous extremity. The hand and arm are wrapped with an elastic bandage, distal to proximal. The hand held by the webril is then suspended overhead. This position drains the edema and maintains shoulder range of motion with the elbow extended.

of the capsule was adherent. He advocated surgical arthrotomy in the following conditions:

1. There is recurrence of adhesion after manipulation
2. The capsulitis is the result of dislocation of the shoulder
3. The capsulitis is the result of fracture of the surgical neck of the humerus
4. X-ray studies reveal bone atrophy (osteoporosis)

Obviously all the indications for arthrotomy are contraindications for manipulation, which he advocated *if* there was none of these contraindications. He "never found success from slow gradual stretching therapy for adhesive capsulitis" (Neviaser).

Turek proposed arthrotomy if

1. There is failure to respond to conservative treatment in regard to both motion and pain (he did not specify whether manipulative therapy was considered conservative)
2. Improvement is too slow to be economically desirable (this would depend upon the occupational needs of the patient)
3. There is recurrence of the pathologic process
4. There is suspected gross damage to the cuff, tendon, and bony structures.

Slow continuous traction (Turek) combined with continuous traction has been used to decrease humeroacromial compression (Fig. 4–10). Slow gradual external rotation is also applied to place the greater tuberosity

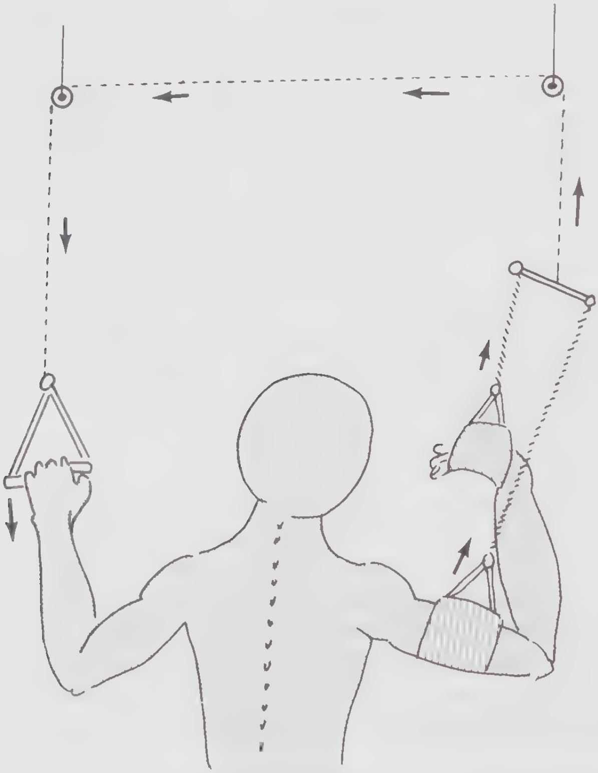


Figure 4–10. Home exercises for painful restricted shoulder. Downward pull by the unaffected arm abducts, elevates, and externally rotates the affected arm.

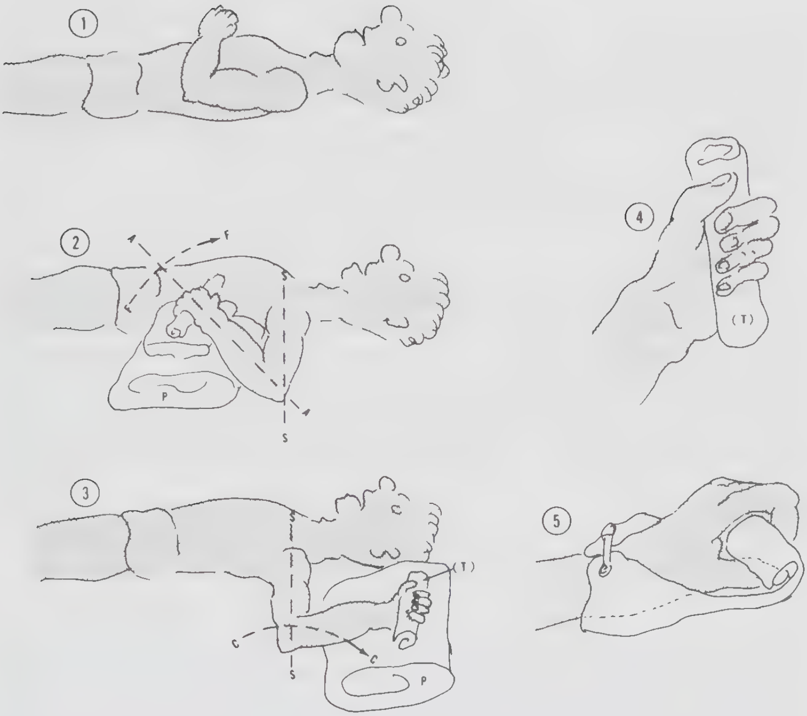


Figure 4–11. Shoulder exercises. With arm abducted to 90° and the hand held with wrist extended (4) and fingers cupped in some extension, the arm is rotated toward overhead position (3). This exercise regains and maintains shoulder range of motion.

behind the acromium gradually. Eventually there is indication for passive (Fig. 4–11) and active exercises to increase flexibility and to regain muscle tone. These have been described in a previous section.

TECHNIQUE OF MANIPULATION

Lundberg claimed that manipulation could significantly improve the range of motion but did not appear to shorten the overall length of aching symptoms significantly. Regaining increased range of motion enhances activities of daily living, which is indeed valuable. The patient to be manipulated should be made aware of the possible gain *and* the possible failure.

Parker and associates expressed the opinion that manipulation under anesthesia should be considered if there was less than 90° of active-passive abduction despite 3 months of aggressive therapy. They use manipulation initially to increase external rotation, followed by abduction, then forward flexion adduction and internal rotation.

Other advocates of manipulation of the frozen shoulder differ in the sequence of manipulation. Hill and Bogumill favor initial abduction, then flexion, and gradually external and internal flexion. The sequence varies according to the findings at the time of examination under an anesthetic and also the experience and expertise of the manipulator. Gentle short arm manipulation is the accepted recommended method.

All advocates of manipulation are strictly in agreement that *no* long lever arm technique should ever be applied. This means that the manipulating arm-hand of the manipulator should be placed upon the humerus *as close as possible to the axilla*, giving a short arm fulcrum upon the glenohumeral joint. During the manipulation the therapist hears *tearing* of adhesions, which implies that this technique is successful from mechanical tearing of the adhesions, either within their substance or at the point of their attachment to the bony periosteum.

DISTENTION ARTHROGRAPHY: BRISEMENT

There are advocates of *breaking the adhesions by arthrographic means.* Normally the glenohumeral capsule will admit up to 30 ml of fluid. In adhesive capsulitis the joint capacity may be decreased to 5 ml or less.

In brisement the arthrogram is performed to determine entry into the capsule and its total capacity. Through the same needle a solution of an anesthetic (to decrease the pain of the procedure) and possibly a steroid (Depo-Medrol—to prevent further inflammation and adhesions) is injected in increasing quantities, attempting to reach an injection of 40 ml. Upon reaching a certain amount, that admitted by the constricted capsule, further injection causes a tearing of the capsule. The manual injection can be only of the force of manually pressing the plunger of the syringe and therefore is not excessively forceful.

The hearing of the tearing also permits immediate increase of range of motion. This testing also adds passive range, mobilization, and even gentle manipulation of the joint in which the capsule is now torn. The objective of gaining increased range thus has been accomplished.

The immediate postinjection treatment is that of manual manipulation. The arm should be held in the abducted, externally rotated overhead position by being tied overhead to the head of the bed.

Arthrotomy technique is beyond the scope of this text. The indications have been given, and once the procedure has been advocated it is the expertise of the orthopedic surgeon that decides the approach and technique. Current studies do not consider surgery by way of the arthroscopic approach to be effective in the frozen shoulder.

BIBLIOGRAPHY

Andren, L and Lundberg, BJ: Treatment of rigid shoulders by joint distention during arthrography. Acta Orthop Scand 36:45, 1965.

Binder, AI, et al: Frozen shoulder: A long term prospective study. Ann Rheum Dis 43:361, 1984.

Bridgman, JF: Periarthritis of the shoulder and diabetes mellitus. Ann Rheum Dis 31:69, 1972.

Cailliet, R: The Shoulder in Hemiplegia. FA Davis, Philadelphia, 1980.

Clark, GR, et al: Preliminary studies in measuring range of motion in normal and painful stiff shoulders. Rheumatology Rehabil 4(1):39, 1975.

Codman, EA: The Shoulder: Thomas Todd, Boston, 1934.

DePalma, AF: Surgery of the Shoulder. JB Lippincott, Philadelphia, 1950.

DePalma, AF, Collery, G, and Bennett, GA: Variational anatomy and degenerative lesions of the shoulder joint. American Academy of Orthopaedic Surgeons, Instructional Course Lectures 6:255, St. Louis, 1949.

Duplay, S: De la peri-arthrite scapulo humerale. Revue Praticien de Trav de Med 53:226, 1906.

Grey, RG: The natural history of "ideopathic" frozen shoulder. J Bone Joint Surg 60A:564, 1978.

Hill, JJ and Bogumill, H: Manipulation in the treatment of frozen shoulder. Orthopedics 2(9):1255, 1988.

Horowitz, MT: Lesions of the supraspinatus tendon and associated structures. Arch Surg 38:990, 1939.

Lee, PN, et al: Periarthritis of the shoulder: Trial of treatments investigated by multivariate analysis. Ann Rheum Dis 33:116, 1974.

Lippmann, RK: The frozen shoulder, periarthritis and bicipital tenosynovitis. Arch Surg 47:283, 1943.

Lundberg, BJ: The frozen shoulder. Acta Orthop Scand (Suppl) 119:1, 1969.

Maitland, GD: Treatment of the glenohumeral joint with passive movement. Physiotherapy 69:3, 1983.

MacNab, I: Rotator cuff tendinitis. In McKibben, B (ed): Recent Advances in Orthopedics. Churchill Livingstone, Edinburgh, 1979.

McLaughlin, HL: Lesions of the musculotendinous cuff of the shoulder. J Bone Joint Surg 26:31, 1944.

Murnaghan, JP: Adhesive capsulitis of the shoulder. Orthopedics 2(1):153, 1988.

Neviaser, JS: Adhesive capsulitis of the pathological findings in periarthritis of the shoulder. J Bone Joint Surg 27:211, 1945.

Neviaser, TJ: Adhesive capsulitis. Orthop Clin North Am 18(30):439, 1987.

Ozaki, J, et al: Recalcitrant chronic adhesive capsulitis of the shoulder: Role of contracture of the coracohumeral ligament and rotator interval in pathogenesis and treatment. J Bone Joint Surg 71A:1511, 1989.

Parker, RD, et al: Frozen shoulder: Part I. Chronology, pathogenesis, clinical picture, and treatment. Orthopedics June 12 (6):869, 1989.

Reeves, B: The natural history of the frozen shoulder syndrome. Scand J Rheumatol 4:193, 1975.

Rizk, TE, et al: Adhesive capsulitis (frozen shoulder): A new approach to its management. Arch Phys Med Rehabil 64:29, 1983.

Simmonds, FA: Shoulder pain with particular reference to the "frozen" shoulder. J Bone Joint Surg 31B:3, Aug 1949, p. 426.

Turek, SJ: The painful and stiff shoulder. Journal of the International College of Surgeons 22(6):695, 1954.

CHAPTER 5

Posture in Shoulder Pain

In the evaluation and treatment of shoulder pain—whether it is a cuff problem, entrapment syndrome, or bursitis—a component of pain in the shoulder girdle is directly influenced by the posture of the patient.

Normally as the patient stands erect, the scapula has a specific alignment with the center of gravity. The glenoid fossa faces upward, outward, and forward. The overhanging acromium is also related in this way to the humeral head, so that as the arm abducts, the greater tuberosity impinges upon the acromial process and the coracoacromial ligament only after 90° or more of abduction.

At the point of arm elevation to 90°, rotation plays a factor in that, when the arm is internally rotated, abduction is decreased by approximately 30°, and when it is externally rotated, elevation is permitted to 120°, because this position permits the greater tuberosity to pass posteriorly to the acromium (see Chapter 1). All these achievable degrees of abduction are appropriate *if* the scapula is in the correct physiologic position.

In the rounded shoulder posture the scapula changes its vertical alignment. The scapula rotates forward and downward, depressing the acromial process and changing the facing of the glenoid fossa (Fig. 5–1).

As a test, in the normal well-postured individual, when in the erect posture both arms can be elevated to the side and ultimately placed over the head. In this ultimate elevated position the arm rests to the side of the head and behind the ears. If this person assumes a rounded posture the arms can no longer abduct and elevate overhead. In this kyphotic postural attitude, the arms can barely reach the ears. Limitation has been *physiologically* limited by merely rounding the trunk posture. No structural tissues have been altered and no pathologic process is present. This is a physiologic, mechanical phenomenon.

Assuming that the person habitually presents with a round back posture—dorsal kyphotic posture—the relationship between the acromium and the abducting humeral head is consistent, and there is encroachment of the greater

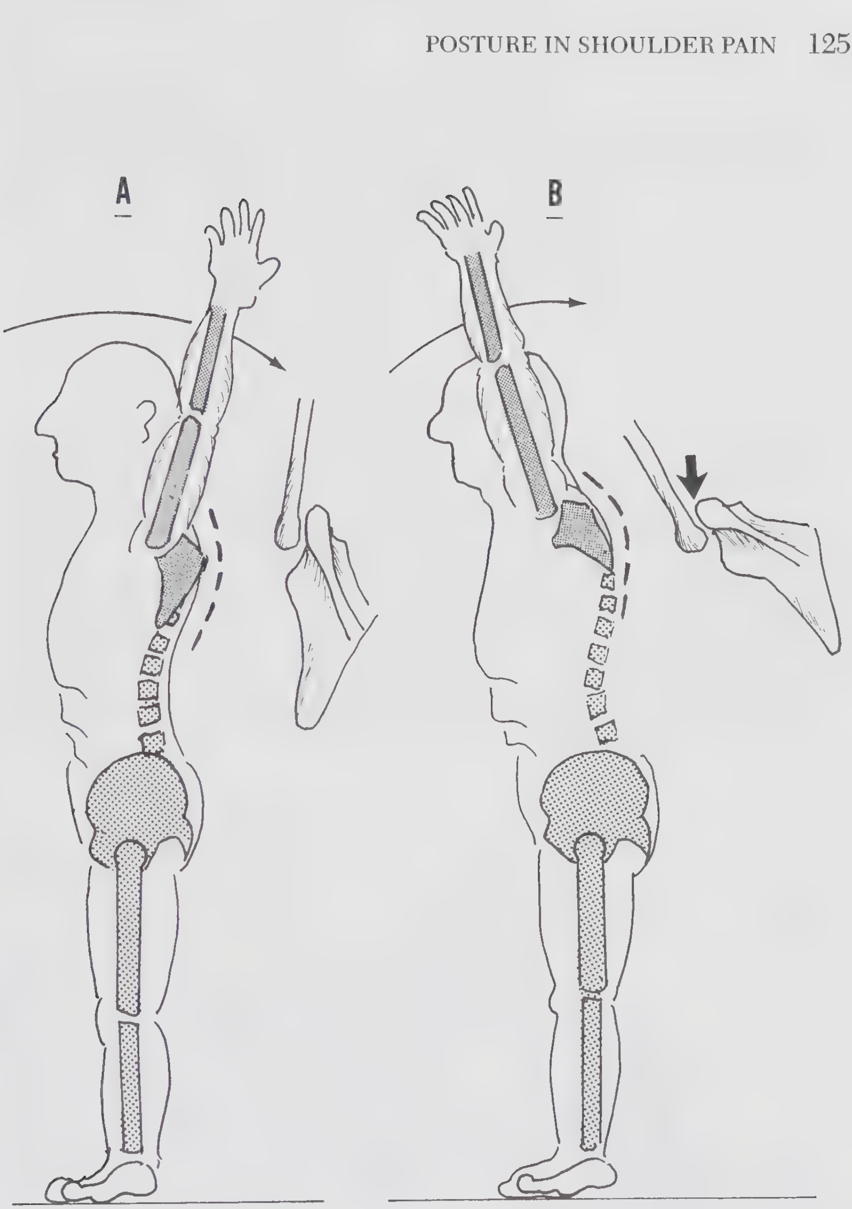


Figure 5–1. The shoulder as related to posture. *(A)* The erect posture with a minimal dorsal kyphosis. The arm, elevated overhead, reaches behind the ear. *(B)* With a rounded-shoulder posture (excessive dorsal kyphosis), the scapula *(insert)* is rotated to bring the overhanging acromium downward. The elevated arm now is mechanically limited in overhead elevation. *(Arrow)* The site of greater tuberosity impingement upon the acromial process.

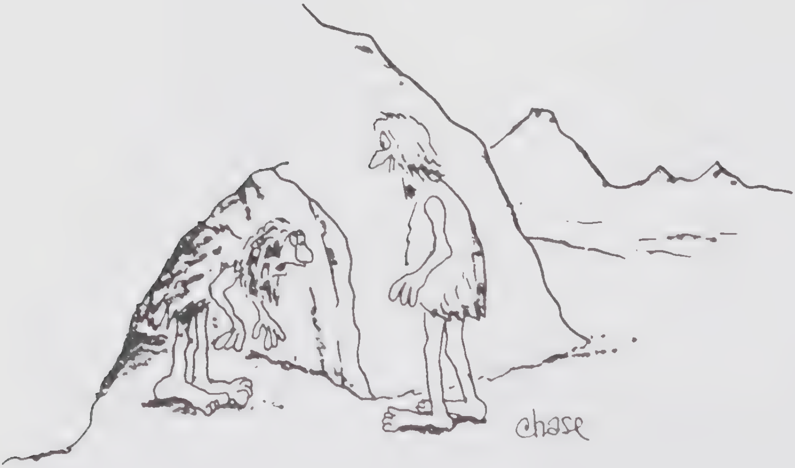
tuberosity and all the tissues contained in the suprahumeral compartment in every arm activity requiring elevation and marked abduction. Entrapment of the rotator cuff is a constant expectation.

Because posture is given a significant role in the cause of shoulder pain as well as in cervical pain (Cailliet), it requires a full discussion. To influence, to alter, or to improve posture, full appreciation of all the factors that affect posture is necessary.

POSTURE

Posture is the attitude humans assume upon standing or sitting in the erect position (Fig. 5–2). Posture has cosmetic and psychologic implications in that we stand the way we feel and project a certain appearance. Posture is also significantly influenced by familial and congenital factors, modified by training and habits. It is influenced by the appearance of others and dictated by occupational demands. It is adversely affected by illnesses of orthopedic or neurologic sequence.

Posture also causes or influences numerous orthopedic and neurologic dis-



"I tried standing erect, but I kept banging my head!"

Figure 5–2. Comic spoof of erect posture.

(From John Chase, reprinted from Science 43:17, March, 1989, with permission of the artist.)

eases or syndromes of pain and impairment as well as shoulder mechanics. Faulty posture augments tissue changes in bony, ligamentous, or muscular structures, and it is believed to adversely affect the spinal column diskogenic tissues. Posture merits thorough evaluation.

POSTURE DEVELOPMENT

The spine of the newborn infant, not yet in an upright posture, who has not reacted to the influences of gravity or assumed an erect position, has none of the adult physiologic curves. The entire newborn spine retains the *in utero* posture, which is that of total flexion (kyphosis). The curvature of the entire newborn spine forms a slightly greater kyphotic curve than the physiologic kyphotic curve of the thoracic spine that will remain throughout life.

The newborn spine has no lordotic curves, either in the cervical or in the lumbar area (Fig. 5–3). The first lordotic curving of the vertebral column is noted in the cervical region during the first 6 or 8 weeks of life. At this stage of development the newborn child extends his or her head from the prone position. This head-neck extension is an antigravity action which occurs by virtue of contraction of the extensor muscles. The action occurs with some propriocep-

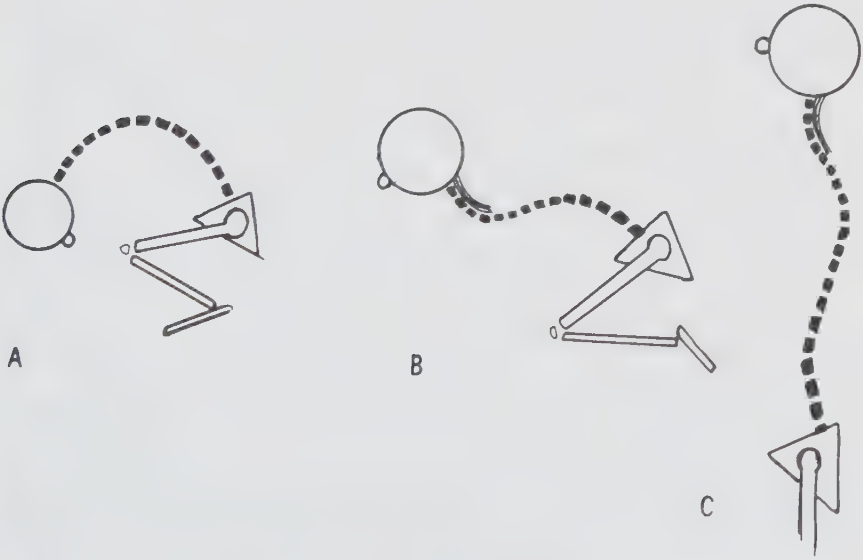


Figure 5–3. Chronologic development of cervical lordosis in the development of posture. *(A)* The curve of the fetal spine *in utero*. *(B)* Formation of the cervical lordosis when the head overcomes gravity. *(C)* Erect adult posture.

tive input and initiation from the basic *righting reflexes.*

The ultimate cervical lordotic curve remains throughout the life of the individual with daily variations from change of position and various activities. The cervical spine is flexible and adheres to the laws of gravity and to muscular action imparted to it. There are numerous factors that modify the degree of curvature of the cervical spine, which will be discussed subsequently.

Because the cervical spine is the uppermost curve and supports the head, it is dependent on the lower spinal column curves: the thoracic, lumbar, and sacral curves.

All the superincumbent curves are flexible and depend upon ligamentous and capsular support and muscular tone to remain erect. The muscular tone is the predominant, albeit not the only, source of support and is the major factor that determines the degree of spinal curvature in relationship to the center of gravity.

The degree of muscular tone is dependent on proprioceptive feedback from the periphery. Proprioceptive impulses ascend from the ground upward through the central nervous system to inform the body of its relationship to the center of gravity (Fig. 5–4).

The proprioceptive *end organs* are located within the skin, the joint capsules, the ligaments, and the muscles of the entire body and are stimulated by pressure variants, movements, and peripheral sensations of touch. In its contact with the ground, the foot receives sensory contact through the skin, the bones, the ligaments, and the joint capsules. There are similar sensations received from the ankles, the knees, the hips, the pelvis, and so on up the spine. All proprioceptive fibers from these end organs send instantaneous information that becomes coordinated in the central nervous system to initiate appropriate muscular tone.

The vestibular system also sends information to this central nervous center to inform the body of its relationship to the center of gravity. These righting reflexes are inherent in the central nervous system at birth and are modified during development as the external and internal situations change.

It becomes apparent that the sensation—here considered *proprioceptive*—is instrumental in maintaining the erect posture and influences the relationship of the erect body to the center of gravity. The sensation also is impressed upon the cortex, which interprets the sensation of the totally erect person.

The sensory motor nervous system, thus, is the major determinant in the process of assuming the erect posture. Because the erect posture is also that of superincumbent curves as they relate to the center of gravity, the degrees of curves are solely dependent on proprioception. The curves are also influenced by appropriate perception of this erect position. Posture is a reflex proprioceptive mechanism that depends also upon cortical appreciation and acceptance.

It is apparent that posture is a neuromuscular reaction to proprioceptive impulses from the periphery and that the *feeling* of that posture as normal or abnormal is a learned process.

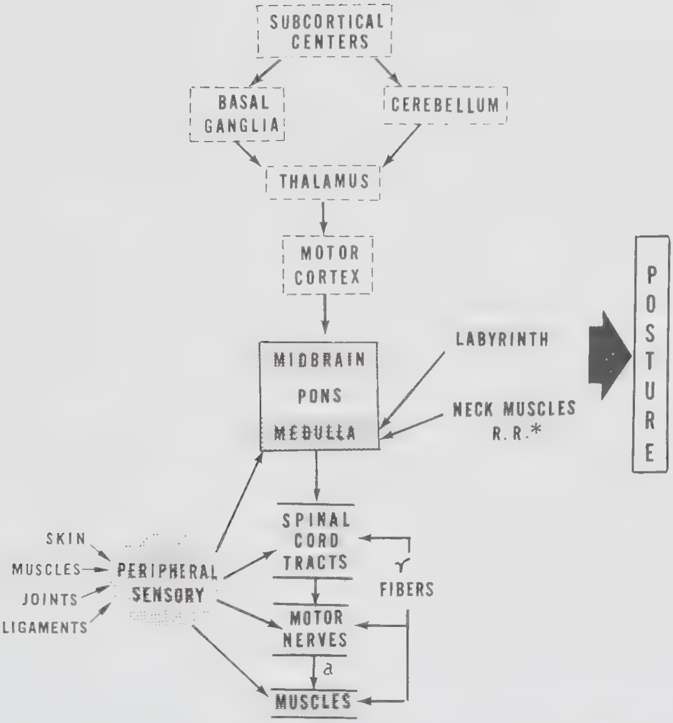

SUBCORTICAL CENTERS
BASAL GANGLIA
CEREBELLUM
THALAMUS
MOTOR CORTEX
MIDBRAIN
PONS
MEDULLA
LABYRINTH
NECK MUSCLES R.R.*
POSTURE
SKIN
MUSCLES
JOINTS
LIGAMENTS
PERIPHERAL SENSORY
SPINAL CORD TRACTS
γ
FIBERS
MOTOR NERVES
a
MUSCLES

Figure 5–4. Neurologic concept of posture. The upper neurologic pathways (subcortical centers, basal ganglia, cerebellum, thalamus, and motor cortex) are the well-documented circuits by which motor activities are initiated and coordinated.

Within the midbrain medulla are the primary righting reflex centers that receive impulses from the labyrinth and the righting reflexes (*R.R.) of the neck muscles. Posture is dependent upon this medullary center and the spinal cord interneurons influencing the motor nerves that are moderated by the alpha and gamma fibers to the muscles.

Each level of the central system receives and is moderated by peripheral sensory impulses from the skin, joints, capsules, ligaments, and muscles (see text).

Upon maturation the thoracic curve becomes less flexible to the extent that there is no further significant flexion or extension in the sagittal motion of the total spine. The thoracic spine of an adult person is essentially *fixed* in its kyphosis. The spinal curves above and below this dorsal kyphosis must, therefore, be correlated in maintaining the correct posture in its relationship to the center of gravity. An increase in the lumbar lordosis accentuates the cervical lordosis, and vice versa.

Admittedly the ultimate spinal curves are initially determined by developmental factors related to gravity. These curves form the erect posture. As a person develops, however, there are at least three major factors that influence the adult posture: (1) heredity, (2) disease, and (3) acquired habit. Of the three, the latter is the factor that is the least well understood, but it is also one that can best be influenced by treatment.

The familial-hereditary factors influencing the posture of the adult patient can be discerned by evaluation of the parents, grandparents, and/or siblings. This *familial* posture is essentially a body type and may be altered slightly, but it is more difficult to modify.

Disease factors influencing posture are too numerous to discuss totally. Examples are inflammatory joint disease such as rheumatoid spondylitis, other rheumatoid disease, neurologic diseases such as parkinsonism, and the effects of structural scoliosis. There are many others that can be ascertained by careful history, examination, and x-ray confirmation. In these cases many aspects of the resultant posture can be modified during the development of the disease if the impact of the disease upon the posture is recognized.

The posture of acquired habit has been considered a neuromuscular phenomenon that has its inception in early childhood. Many factors affecting the growing child play a role. Feldenkrais has thoroughly expounded this concept.

The feeling of proper erect posture develops as a person's nervous patterns develop. Proprioception ascends from the ground up to the neck. The head assumes a *normal* balance on the neck when there is comfort and ease of remaining erect. All proprioception is subconscious yet constantly bombards the righting reflexes to maintain erect posture.

There is little or no need to be aware of all the components of the body involved in the erect posture. The total posture is felt to be proper. This concept implies that when all proprioception influencing the muscles results in the erect spine, the person feels that the posture is normal, that is, correct, proper, effortless, cosmetically proper, and pain free.

The erect stance and body position *feels* like a proper posture. The vestibular apparatus within the head also imparts proprioception which, when considered to be a proper posture by the feeling, asserts no effort to modify the righting reflexes.

Whatever modifies or influences the resultant reflex action yet imparts the feeling of normal or correct affects the posture. What are some of these factors that lay down the neuromuscular patterns? What influences the proprioceptions? More important, what remains when the pattern is considered normal by feel but is structurally abnormal and undesirable?

Bad habit develops in childhood. The slumped-over posture is seen often in adolescents and young adults. The posture assumed becomes such a daily occurrence that it becomes normal; that is, it *feels normal*. This slumped posture may be the result of family or peer pressures—the result of anxiety, insecurity, fear, anger, or despondency in early childhood. Daily activities with their resultant postures may also influence this posture. Feldenkrais has postulated that many postures develop from the "cringing from fear of physical assault by domineering parents or siblings."

As the years go on, the assumed posture becomes comfortable and accepted as normal. The proprioceptive stimuli of this assumed posture have no impact upon the cortical interpretation. The posture (here the forward-head posture) is accepted, and the muscular tone needed to support this posture is also accepted. There is no perceived need for the person to correct the assumed posture inasmuch as there is no discomfort or fatigue initially experienced.

The fact that the posture holding the head *ahead* of the center of gravity is physiologically incorrect (Fig. 5–5) and demands excessive muscular action is ignored or, at best, not perceived.

Unconscious habits develop in adolescence, such as in the young woman whose breasts are larger than those of her companions or in the person who feels he or she is too tall, when the person may assume a slumped-forward posture to be less *different*. The persistence of the assumed posture for many years becomes normal and persists long into life when the original reason is no longer pertinent or recognized. The pattern now is well established not only in the neuromuscular complex but also in the musculoskeletal system.

A daily exercise can be instituted to correct faulty posture (Fig. 5–6). Admittedly, daily observation of proper posture must assure that posture is constantly erect and not only during the few minutes of doing posture exercise. Done frequently, the exercise implements the correct position, strengthens the short neck flexors, stretches the tight neck extensors, gives the feeling of pushing up to a taller stance, and essentially decreases the cervical lordosis.

As the lordosis decreases, the head becomes centered above the center of gravity. This is accomplished, in the exercise, by gliding the head back while keeping the chin level. This, termed a *translation* exercise, causes an anteroposterior shear effect. Once the tissues (ligaments, muscles, fascia, and joint capsules) responsible for proper posture are well conditioned, the feeling of proper erect posture becomes more automatic.

As the cervical lordosis decreases and the head assumes a central position over the center of gravity, the shoulder blades simultaneously assume their correct position upon the thoracic wall without their being singularly considered.

Acquired posture can also be influenced by daily activities that demand a forward-head posture. Job requirements such as operating a computer and viewing a terminal screen demand a forward-head posture. Faulty vision or its correction by bifocal or trifocal spectacles may place demands on the posture that are undesirable. All these causative factors demand a careful study of the activities of daily living to determine their effects on the posture.

Emotions also influence posture. We stand, sit, and walk *as we feel.* Body language is well recognized. The depressed person walks, stands, and sits in a depressed manner. The angry person personifies his or her anger in the body stance. The impatient individual depicts the impatience in posture as well as in actions. The study of body language is widely discussed and is recognized by the astute practitioner. Its effect on the undesirable posture must be determined and addressed.

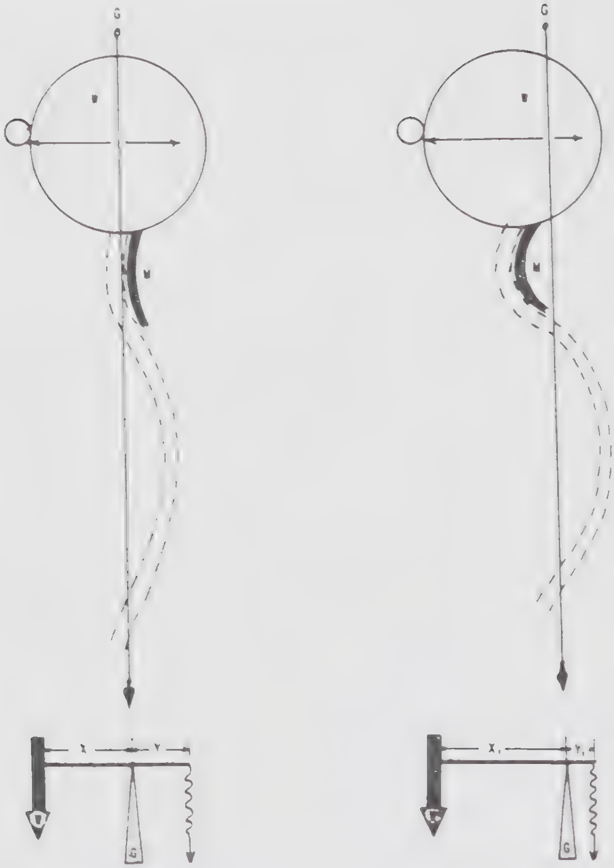


Figure 5–5. Gravity effect on a forward-head posture with increased lordosis

W = Weight of the head. Remains constant.
X = Distance of head weight (W) from center of gravity (G).
Y = Distance of spinal musculature from center of gravity (G).
M = Tension developed by musculature to sustain weight of head (W)

$$W \times X = M \times Y$$

When there has been a painful shoulder with the painful arc on abduction overhead elevation, indicating entrapment of the greater tuberosity on the acromial process and the coracoacromial ligament, the contributory posture must be evaluated. If all components of treatment of the painful shoulder are instituted and relatively effective, to allow the incriminating postural component to persist is to allow ultimate recurrence of the painful shoulder.

The psychologic aspect of the postural component may also indicate to the

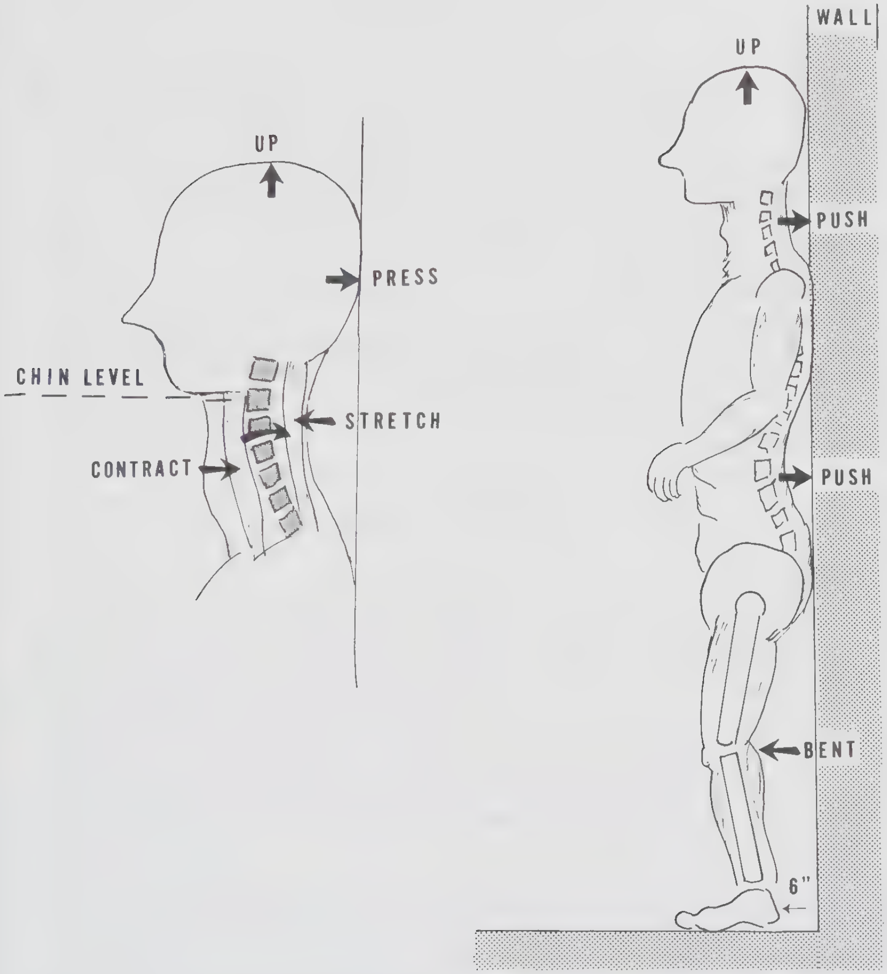


Figure 5–6. Proper posture exercise. Standing 6 inches away from a wall with the knees slightly bent, the postural attitude is *standing tall* (UP). The head is pressed against the wall. The chin is kept level and the neck is pressed against the wall *(curved arrow)*. This translation motion contracts the neck flexors and stretches the neck extensors. All motions are done simultaneously, repeatedly, and slowly. At first the effect may be a slight *strain,* but ultimately the resultant posture is accepted and automatic. This exercise can gradually be done without the wall, in the sitting posture as well as while standing.

examiner that the neuromusculoskeletal aspect of shoulder function is also affected. Both must be addressed for ultimate success in pain relief and treatment of degenerative tissue sequelae.

BIBLIOGRAPHY

Body, IA, et al: The role of the gamma system in movement and posture. Association for the Aid of Crippled Children, 345 East 46th Street, New York, 10017, 1964.

Brunnstrom, S: Clinical Kinesiology, ed 3. FA Davis, Philadelphia, 1972.

Burke, D and Eklund, G: Muscle spindle activity in man during standing. Acta Physiol Scand 100:187, 1977.

Cailliet, R: Neck and Arm Pain, ed 3. FA Davis, Philadelphia, 1991.

Ertekin, N and Ertekin, C: Erector spinae muscle responses while standing. J Neurol Neurosurg Psychiatry 44:73, 1981.

Feldenkrais, M: Body and Mature Behavior: A Study of Anxiety, Sex, Gravitation, and Learning. International Universities Press, Inc., New York, 1949.

Joseph, J: Man's Posture: Electromyographic Studies. Charles C Thomas, Springfield, IL, 1960.

Kelton, IW and Wright, RD: The mechanism of easy standing by man. Australian Journal of Experimental Biology and Medical Science 27:505, 1949.

Lovett, RW: Lateral curvature of the spine and round shoulders. P Blakiston & Son, Philadelphia, 1907.

Roaf, R: Posture. Academic Press, New York, 1977.

CHAPTER 6

Trauma to the Shoulder

DISLOCATION

The shoulder is unique in its wide range of motion when compared with other joints in the body. The bony restraints are minimal, but soft tissues constrain motion. These include the glenohumeral ligaments, which are thickenings of the glenohumeral capsule and the musculature of the joint. Injury to the shoulder thus involves these soft tissues.

Of the numerous traumas to the shoulder, probably the most painful and disabling is the dislocation. The initial dislocation is usually an acute trauma, whereas the recurrent dislocation may be a lesser and often unexpected incidence.

Of shoulder dislocations, 90 percent occur anteriorly, which is attributed to the fact that there is anatomic weakness of the anteroinferior capsule through which the humeral head dislocates.

The glenohumeral capsule is thin and loose and is reinforced anteriorly by folds that are called *glenohumeral ligaments*. These ligaments attach at the humerus and fan out to attach to the superoanterior aspect of the glenoid fossa and partly to the glenoid labrum, with a small portion attaching to the scapula itself. An opening frequently is found between the superior and middle glenohumeral ligaments termed the *foramen of Weitbrecht* (Fig. 6–1). This foramen may be a frank perforation, or it may be covered by a thin layer of capsule. The articular cavity connects with the subscapular fossa through this opening and it is through this opening that the humeral head usually dislocates when trauma occurs to herniate the head of the humerus out of the capsular confines. Recurrent herniation may occur when there has been fraying or actual destruction of the middle glenohumeral ligament.

Primary anterior dislocation occurs from trauma at any age, but recurrence of dislocation is highest in the young (teens to 20s) and decreases after age 45,

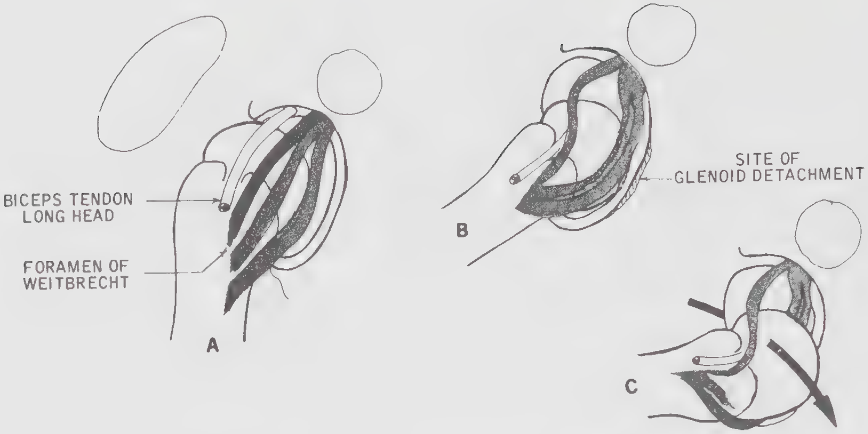


Figure 6–1. Anterior capsule, glenohumeral ligaments, and avenue of anterior shoulder dislocation. *(A)* The three folds of the anterior capsule forming the glenohumeral ligaments that attach from the anterior ridge of the humerus to the glenoid fossa. *(B)* A tear is shown in the area between the superior and middle ligament (foramen of Weitbrecht), which may be covered by a thin capsule or may be a direct opening. *(C)* As the head of the humerus moves forward and downward, it emerges through the opening.

possibly because physical activities decrease after age 45. Also, there is greater fibrous tissue repair in later life. In an acute dislocation with severe hemorrhaging, greater resultant scar formation may prevent possibility of recurrent dislocation.

In any dislocation there may occur partial or complete detachment of the glenoid labrum at the anteroinferior aspect. The concept of the labrum varies as to that of fibrocartilage or a redundant folding of the anterior capsule, but regardless of structure, trauma to the labrum can avulse the head of the humerus as opposed to a tear within the capsule between the ligaments. Normally a *pouch* is noted in the capsule where it allegedly folds over to form the labrum which disappears when the arm is internally rotated. This pouch presents a potential site of dislocation of the humeral head.

There are four types of dislocation (Fig. 6–2), the most common being the subcoracoid. The type of dislocation is termed according to the ultimate site of the humeral head in relation to the glenoid fossa when the diagnosis is made. Less common are the subclavicular and subglenoid dislocations. The posterior dislocation, the subspinous, is rare. These latter types may be a progression of the initial subcoracoid, inasmuch as any anterior dislocation may change into any of the other three types of dislocation.

Occult subluxation of the shoulder may be missed by the examiner unless it is suspected (Glousman and Jobe), especially in athletes who use overhead

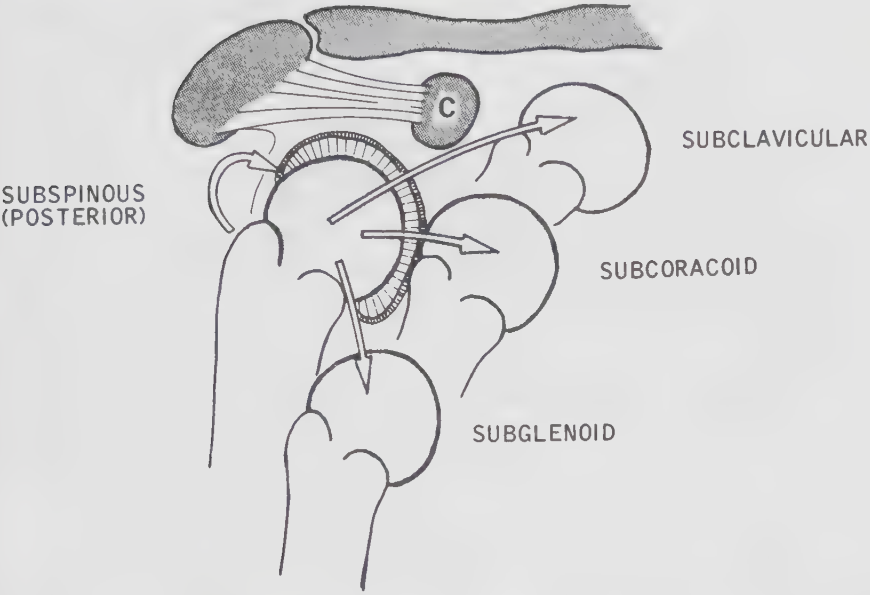


Figure 6–2. Four types of shoulder dislocation. The subcoracoid dislocation is the most frequent, and the subspinous (posterior), the least common. All three anterior dislocations—the subcoracoid, the subglenoid, and the subclavicular—may alternate. The type designated depends upon the position of the humeral head in relation to the glenoid seat at the time of diagnosis.

motions (see Chapter 7, Athletic Injuries). Frank dislocation or recurrent dislocation may be obvious.

Mechanism of Dislocation

Trauma is the usual cause of dislocation. The offending trauma is usually an incidence that *catches* the muscle unprepared or is of sufficient force that it overwhelms the resisting musculature, which is inadequate in the anteroinferior fold aspect of the capsule. The musculature—anteriorly the pectoralis major and posteriorly the latissimus dorsi—does not significantly protect the inferior capsule.

Young athletes whose sport involves frequent and forceful overhead motions may develop occult subluxation with merely pain upon performing the activity but no clear evidence of joint instability. In these cases, although not causing symptoms of joint instability, there is a sensation of weakness, with the arm *going dead* (paresthesia) during the activity, in addition to pain upon performance. These symptoms probably indicate traction on the brachial plexus during the activity, which would not occur in the presence of an adequate capsule.

In the young, the posterior tissues, the supra- and infraspinatus tendons, which attach to the greater tuberosity, are intact and the anterior capsule is thin and loose, therefore dislocation occurs anteriorly (Fig. 6–3). In older people the posterior tissues are less flexible and tear on the greater tuberosity avulses allowing the head of the humerus to "roll over" the anterior rim of the glenoid fossa.

The frequency of anterior dislocation, depicted in Figure 6–4, was explained by Codman as the ability of the arm to rotate being diminished in downward movement because in the overhead position, the arm is locked in a fixed position against the acromial process. This prevents rotation. To fully elevate the arm overhead in the coronal plane (Fig. 6–5), the humerus must be fully externally rotated.

This places the greater tuberosity against the overhanging acromium. Descent of the arm must include internal rotation. If either of these rotational ad-

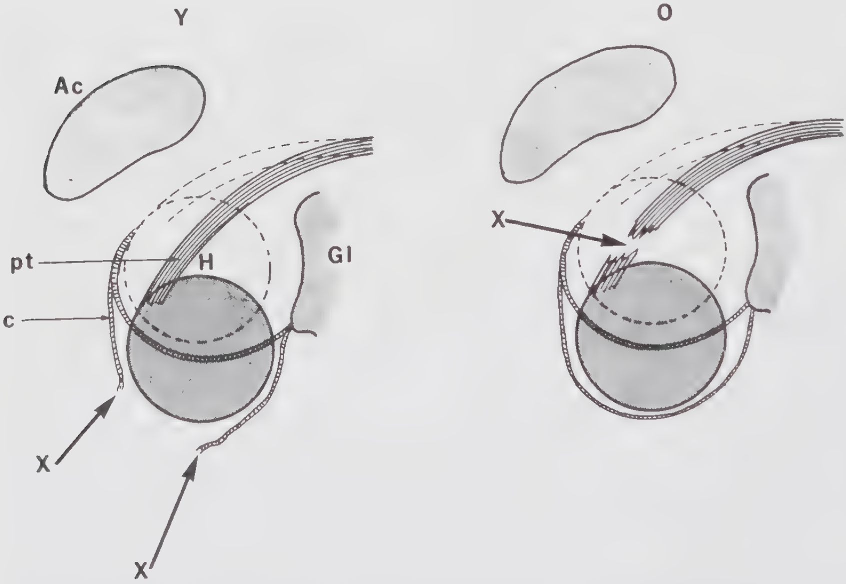


Figure 6–3. Relationship of age of patient to mechanism of anterior dislocation. Y shows the normal relationship, in the young, of the head of the humerus (H) to the glenoid fossa (GI) with intact posterior tissues (pt) consisting of the supraspinatus and infraspinatus tendons attached to the greater tuberosity. The capsule (c) is intact, albeit thin and loose. The mechanism of dislocation in the young causes tear through the anterior capsule (X *arrows*). (*O*) In older people, the posterior tissues tear (X *arrow*), or the greater tuberosity avulses. In this latter mechanism the head of the humerus *rolls* over the anterior rim of the glenoid but does not tear the anterior capsule.

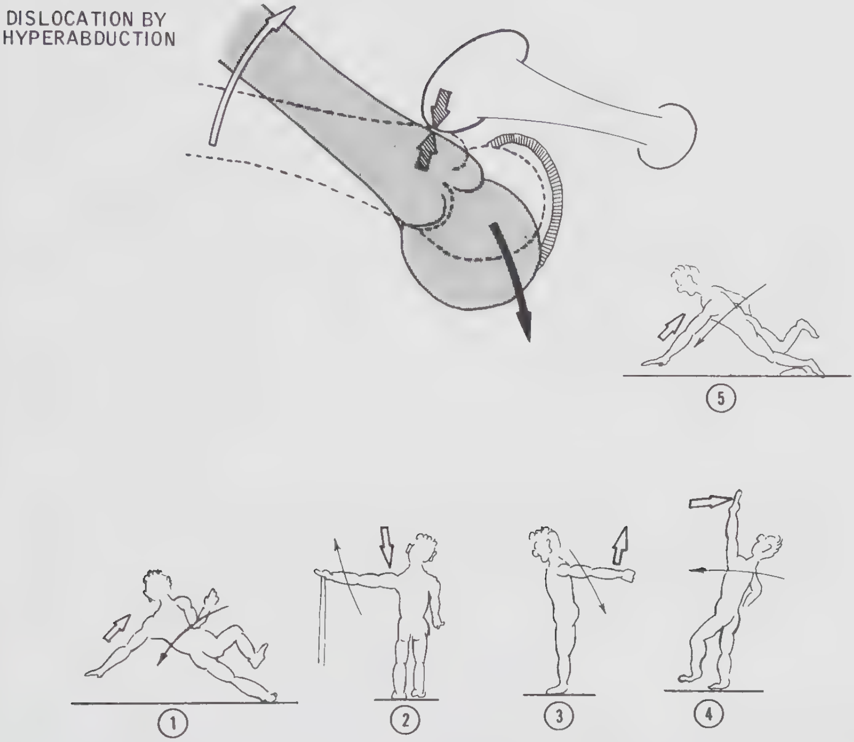


Figure 6–4. Mechanism of dislocation: *hyperextension theory*. Abduction with the humerus in internal rotation or forward flexion with the arm in external rotation becomes limited by the acromial arch. Forceful elevation when this point of impingement has been reached uses the arch as a fulcrum and dislocates the proximal head by causing it to descend and move forward.

justments are violated and force is applied to accomplish elevation and descent of the arm, a fulcrum is created. The distal force, now with a long lever arm, causes downward and anterior displacement of the proximal head of the humerus. All that holds the head of the humerus is the inferior capsule, which is inadequate to prevent subluxation or frank dislocation.

The past literature differed in regard to the mechanism of primary dislocation as compared with that of recurrent dislocation, which was attributed to improper reduction. Recurrent dislocations are currently attributed to abnormal laxity of the capsule and to weakness of the surrounding muscles (albeit denied by Bankhart). Abnormal laxity of the capsule is believed to result from excessive stretching or imperfect healing after reduction; muscular weakness, from inadequate postreduction rehabilitation.

There are other factors that have been overlooked, such as glenoid frac-

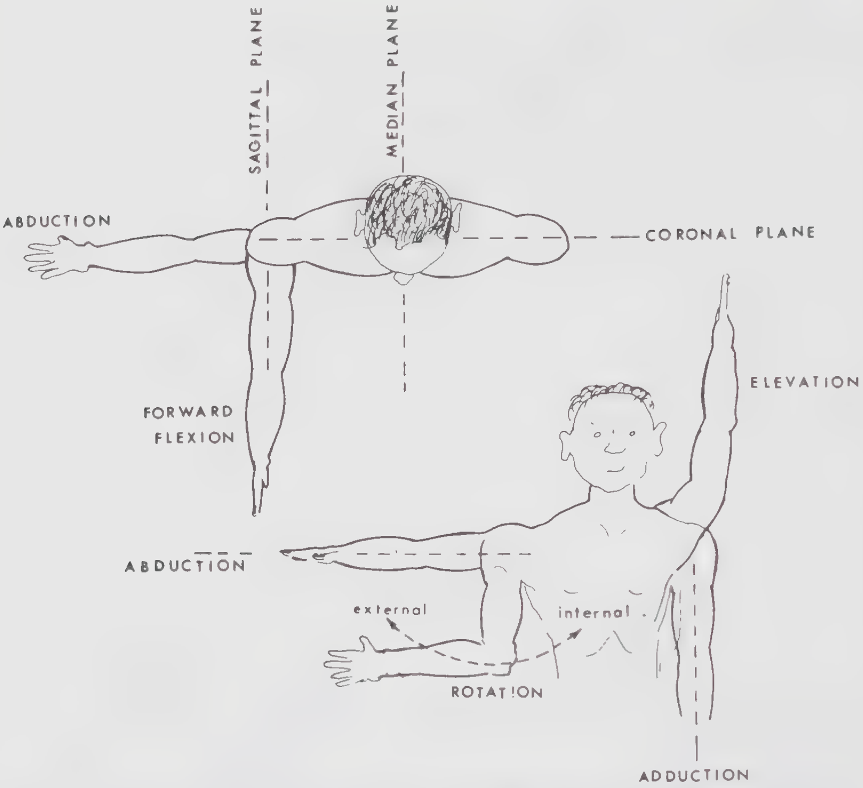

SAGITTAL PLANE
MEDIAN PLANE
ABDUCTION
CORONAL PLANE
ELEVATION
FORWARD FLEXION
ABDUCTION
external
internal
ROTATION
ADDUCTION

Figure 6–5. The planes of arm movement, indicating the direction of movement and the planes of movement in relation to the body. The body is viewed from above and from the front. All arm planes are related to these two body positions.

tures (Fig. 6–6), labrum tears, fractures of tuberosities, deformation of the humeral head, and contractures of some of the muscles about the joint. Better radiologic studies, such as computerized tomography (CT) scanning and MRI studies as well as arthroscopic investigation now reveal these defects more frequently.

Fragmentation of the anterior bony ridge or a posterior lateral defect has been discussed in the literature as the *compression notch of Hermodsson* (Fig. 6–6). Special x-ray studies are needed to reveal this defect, and thus it must be suspected in the case of recurrent dislocation. This fracture of the glenoid rim is attributed to pivoting around a strong unyielding coracohumeral ligament, causing compression of the posterior lateral portion of the humeral head against the sharp posterior rim of the glenoid fossa. A compression defect results.

Admittedly rare, posterior dislocation does occur and must be recognized

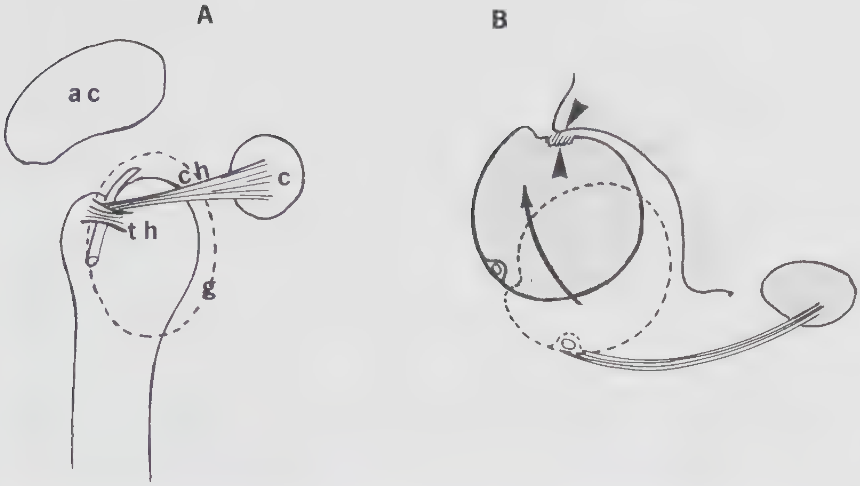


Figure 6–6. Intra-articular fracture in recurrent shoulder dislocation *(notch lesion of Hermodsson).* A strong coracohumeral ligament acts as a pivot point that causes the humerus to compress against the posterior rim of the glenoid and to cause a compression fracture (indenture) in the posterolateral aspect of the humerus.

early, otherwise reduction becomes difficult and postdislocation complications are frequent. In a posterior dislocation the head of the humerus ultimately resides *behind* the scapula—a result of direct trauma from the front to the arm that is in a flexed and abducted position. Posterior dislocation has also occasionally been noted after a convulsive seizure.

Diagnosis of Dislocation

In the occult subluxation of the young athlete there is a history of paresthesia of the upper extremity during the activity. The arm goes *numb*, and there is subjective weakness. This has been attributed to probable brachial plexus traction during the activity. If there has been an occult posterior subluxation, the posterior movement of the arm occurs during an athletic push-up or while performing a bench press against weights. The presence of subluxation is revealed by a thorough clinical examination with confirmation by ancillary tests, even including an arthroscopic examination. The importance here is the suspicion of a subluxation in a young athlete with bizarre symptoms but no overt evidence.

In the anterior dislocation, because the humeral head is not in its usual position, the appearance of the shoulder is abnormal. The normal rounded appearance of the shoulder is lost when compared to the opposite side. The acromium appears unusually prominent because of the hollow space below the acromium where the humerus usually presents.

All movements, active and passive, are limited and painful. Because the head of the humerus is *locked* in a medial position, the elbow sticks out away from the body. If the dislocation progresses to a subglenoid position, the arm may become locked in this full abducted position; termed *luxatio erecta*. This position diagnostically depicts a subglenoid dislocation.

In posterior subluxation the arm becomes fixed in internal rotation, and any attempt at passive or active external rotation is resisted and usually impossible. Even regaining a neutral position is prevented. The coracoid process is prominent, and frequently the humerus can be palpated posteriorly under the spine of the scapula.

Proper x-ray studies are diagnostic, but a word of caution must be expressed: *Routine* x-ray studies may be deceivingly *negative*. Axillary and tangential views are necessary and must be ordered when dislocation is clinically suspected.

Persistent pain after reduction may indicate glenoid fracture, avulsion fracture of the greater tuberosity, and rotator cuff tearing. Appropriate examination and confirmative studies, described in previous chapters, are needed.

Treatment of Dislocation

Closed reduction of a shoulder dislocation is usually possible without surgical intervention. Anesthesia may be necessary. The usual position of the patient for closed reduction is prone on a table with the arm dangling—the original method of Hippocrates and still considered effective.

Gentle traction upon the arm is frequently needed as well as specific manuevers. The surgeon's stocking foot is placed in the axilla for counterpressure, and traction is gradually applied. While traction is applied, the arm is brought in toward the body, levering the head of the humerus around the fulcrum now presented by the surgeon's foot.

The longtime method of Kocher (Fig. 6–7) is still advocated but needs skill and gentleness. The *click* elicited in this manuever may be interpreted as the accomplished reduction when it may actually mean a change from anterior subluxation to another type of dislocation. The external phase of the manuever may tear the subscapularis muscle insertion or even cause a spiral fracture of the humerus if done improperly, forcefully, or on an elderly osteoporotic individual.

Upon reduction the arm is immobilized to the anterior chest wall in a sling support to prevent external rotation and should be held there for 3 weeks. During the 3 weeks of immobilization the wrist, elbow, and fingers must be actively exercised frequently. After 3 weeks the splint is removed, and active exercises begin to strengthen adduction, internal rotation, and abduction in the internally rotated position. No active assisted exercises and no passive stretching exercises

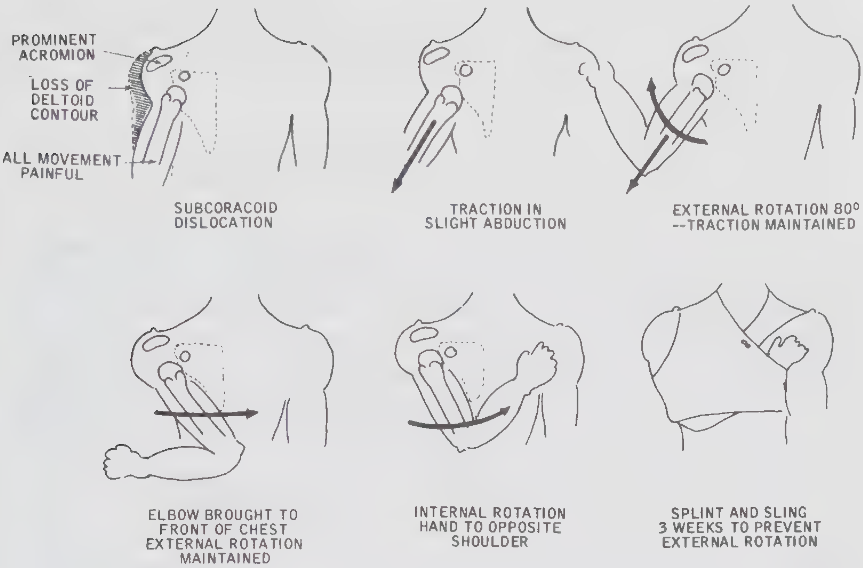

PROMINENT ACROMION
LOSS OF DELTOID CONTOUR
ALL MOVEMENT PAINFUL
SUBCORACOID DISLOCATION
TRACTION IN SLIGHT ABDUCTION
EXTERNAL ROTATION 80°
--TRACTION MAINTAINED
ELBOW BROUGHT TO FRONT OF CHEST
EXTERNAL ROTATION MAINTAINED
INTERNAL ROTATION HAND TO OPPOSITE SHOULDER
SPLINT AND SLING 3 WEEKS TO PREVENT EXTERNAL ROTATION

Figure 6–7. Kocher manipulation for closed treatment of dislocation. All movements should be done smoothly and gently. Traction should be maintained constantly. Once reduced, the arm is splinted for 3 weeks to prevent external rotation.

should be allowed. Isometric exercises here are of value and should be prescribed, properly described, and supervised.

If an avulsion of the supraspinatus muscle tendon or an avulsion of the greater tuberosity has occurred, the arm must be splinted in an abducted externally rotated and preferably forward flexed position. This position obviously diminishes the tension on the supraspinatus muscle tendon and the greater tuberosity and may be maintained in an airplane splint.

Reduction of a posterior dislocation is usually achieved under an anesthetic. Gentle traction in the line of the humerus with simultaneous gentle pressure exerted behind the head of the humerus will usually bring the head of the humerus into the cradle of the glenoid fossa. It may be necessary to apply external rotation to the humerus during reduction. Once reduced, the arm is splinted in a plaster cast in external rotation and with slight abduction, elbow behind the midline of the trunk. This position keeps the humerus ahead of the glenoid fossa level. After 3 weeks, active exercises are begun. There is *no indication* for any passive stretching. The stress of exercises is directed toward external and abduction strengthening. Full range of motion is usually gained within 6 weeks.

Open reduction is indicated in old dislocations, that is, unreduced or recognized dislocations, cuff tears, avulsions, or in severe glenoid changes.

In older people who suffer an anterior dislocation the tearing of posterior structures (see Fig. 6–3) may prolong the convalescence, and prolonged stiffness, pain, and disability may result.

ACROMIOCLAVICULAR JOINT INJURY

In numerous sports activities direct injury frequently occurs to the acromioclavicular joint. These injuries are usually the result of direct superior or lateral blow to the shoulder or a fall on the extended arm (Thorndike and Quigley, Poirer and Rieffel). The blow causes the scapula to move downward until the clavicle impinges upon the rib cage and can move no more. The brunt of the stress then is borne by the acromioclavicular joint and may cause a tearing of the capsular fibers with resultant separation or a strain-sprain injury wherein there remains adequate fibers within the capsule (Fig. 6–8).

Diagnosis

Upon eliciting a history of trauma, there is subjective pain and tenderness in the region of the acromioclavicular joint. Movement evoking pain is diagnostic. Elevation and circumduction of the scapula without simultaneous gleno-

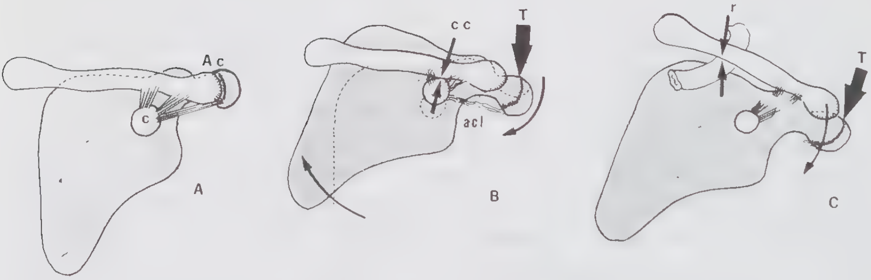


Figure 6–8. Acromioclavicular injuries. *(A)* The normal shoulder with the acromioclavicular joint (A–C) shown. The coracoid (c) is the point of attachment of the coracoclavicular ligaments (cc) and the coracoacromial ligament (acl). *(B)* Indicates that trauma (T) from above can cause luxation of the acromioclavicular joint without ligamentous disruption but with downward rotation of the scapula. *(C)* With excessive force T the clavicle impinges upon the first rib *(small arrows)*. There is additional downward motion of the scapula that further subluxes the acromioclavicular joint but tears all the ligaments. The claviculoscapular joint is now unstable.

humeral motion elicits pain in the acromioclavicular region. This movement can actually be refused by the injured patient.

Abduction forward flexion of the entire arm may evoke pain but usually not until the scapular phase of upper extremity motion occurs. It must be remembered that the scapula remains relatively fixed during early arm abduction, then begins rotation about the acromioclavicular joint as further abduction and overhead arm motion occurs.

Dislocation of the acromioclavicular joints produces a characteristic deformity known as a *shoulder pointer.* This can be noted by the examiner when viewing both shoulders of the undressed patient. Significant dislocation permits the scapula to fall away from the clavicle, and the acromium lies *below and in front* of the clavicle. Upon being palpated the clavicle assumes an unusual prominence, and when palpating the clavicle towards the outer edge the examiner will note that there is a *step down* upon approaching the acromium.

If there is merely an injury where the fibers are sprained or partially torn, a first-degree injury is claimed. There is local tenderness but no instability. A second-degree injury is claimed when there have been sufficient fibers torn to allow some subluxation but stability remains. This can be verified by stress x-ray studies with a 10-pound weight held by the dependent arm. These injuries usually recover without surgical intervention.

Complete tearing of the acromioclavicular capsule fibers results in complete separation, verified by x-ray studies and the weighted arm technique. These injuries are the ones most treated by surgical repair, although many orthopedic surgeons advise conservative treatment as long as the conoid and trapezoid ligaments remain intact (Gurd, Moseley, Urist).

Because there is a meniscus within the acromioclavicular joint (see Chapter 1), damage to the joint often results in posttraumatic arthritic changes. In this condition there is

1. Local pain and tenderness over the acromioclavicular joint
2. Aggravation of pain and tenderness with crepitation on shoulder elevation, circumduction, and anteroposterior motion (without glenohumeral joint motion)
3. Pain and tenderness in the acromioclavicular joint may be aggravated by arm abduction and overhead elevation above 90° abduction
4. There is temporary relief by local injection of an anesthetic agent into the joint

Treatment of first- and second-degree injuries is

1. Immobilization with arm elevation and clavicular depression to approximate the acromioclavicular joint. Simple strapping (Fig. 6–9) must be applied carefully. The limb is encircled to depress the clavicle above and to elevate the arm below. This approximates the acromioclavicular joint.

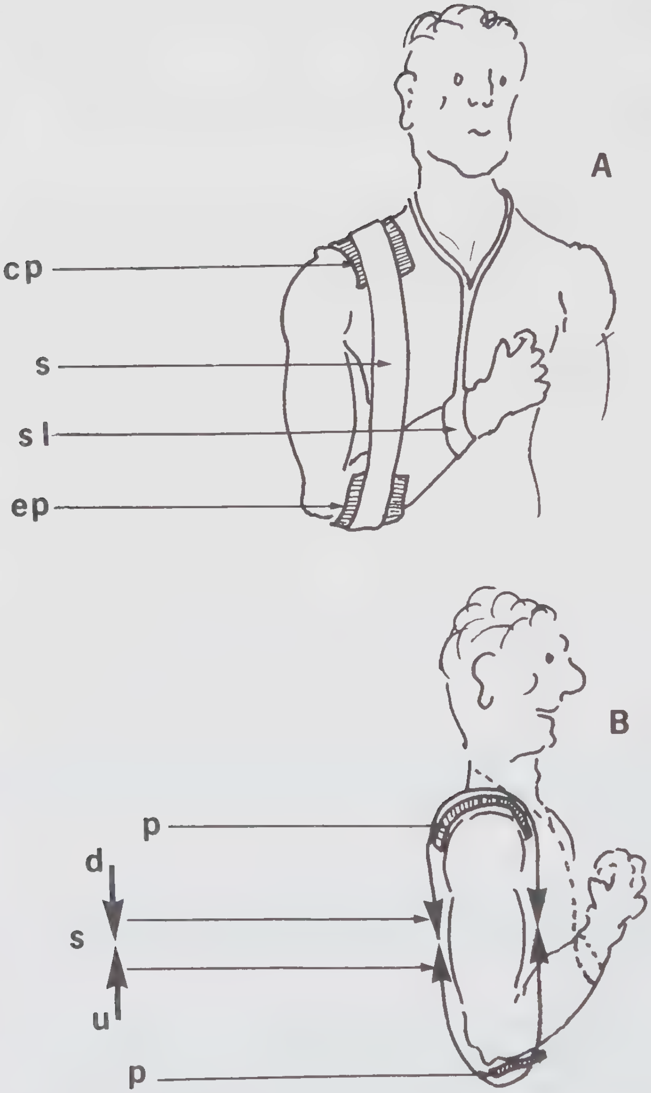


Figure 6–9. Immobilization of acromioclavicular separation: strapping. *(A)* The anterior view in which a circular sling presses down on the clavicle and elevates the arm. A pad protects the clavicle (cp) and the elbow (ep). The wrist is held by a simple sling around the neck (sl). *(B)* The lateral view shows the direction of pull from the circular strap: down upon the clavicle (d), and elevation of the arm (u). If there is no sling, the weight of the arm pulls the acromial fragment down, and the muscles elevate the clavicle, thus separating the acromioclavicular joint. The pads protect the nerves in the shoulder and elbow regions.

Pads are placed to minimize peripheral nerve pressure at the elbow and the axilla. Immobilization may require merely 10 days in a minor sprain. In subluxation 3 weeks are indicated, and in complete subluxation 6 weeks are necessary.

2. Local anesthetic agent injected directly into the joint.
3. Internal fixation in third-degree injuries with significant pain and impairment. If there remains chronic unremitting pain, resection of the outer one-half to one-third of the clavicle may be indicated (acromioclavicular arthroplasty).

There remains controversy as to the best management of acute acromioclavicular dislocation: surgical or nonsurgical. Review of 54 patients (Bannister and associates) indicated that patients treated nonsurgically returned to their work or sports sooner than those undergoing surgery and that after 4 years had excellent to good results in regard to joint range of motion, limitation, and pain.

Surgery, which consists of cancellous or malleolus screw, excision of the meniscus, and repair of the deltoid ligament (Bannister and others), should, however, be considered when there is a displacement of the distal clavicle of greater than 2 cm.

CLAVICULAR FRACTURES

Inasmuch as this is not a complete orthopedic text dealing with numerous fractures, most fractures are not discussed. A Neer type II fracture of the lateral third of the clavicle, however, does present itself in numerous types of injuries similar to acromioclavicular joint injuries and so merits discussion here from the primary physician's perspective.

Neer type II distal fractures, also termed *interligamentous*, are rare (making up only 15 percent of all clavicular fractures), but they appear to be complicated more often with nonunion fractures, hence they encourage open reduction.

Closed reductions has yielded satisfactory results (Deafenbaugh and colleagues)—even superior results to those of nonsurgical reduction of grade III acromioclavicular separations (Taft and associates). Treatment is immobilization with a Kenny Howard sling, followed by postimmobilization strengthening exercises.

BIBLIOGRAPHY

Adams, JC: Review of 180 cases of recurrent dislocation of the shoulder. J Bone Joint Surg 30(B):26, 1948.

Allman, FL: Fractures and ligamentous injuries of the clavicle and its articulations. J Bone Joint

Surg 49A:774, 1967.

Bankhart, ASB: Recurrent or habitual dislocation of the shoulder. Br Med J 2(11):32, 1923.

Bannister, GC, et al: The management of acute acromioclavicular dislocation. J Bone Joint Surg 71B:848, 1989.

Codman, EA: The Shoulder. Thomas Todd, Boston, 1934.

Deafenbaugh, MK, et al: Nonoperative treatment of Neer type II distal clavicular fracture: A prospective study. Contemp Orthop 20 (4):405, 1990.

DePalma, AF: Recurrent dislocation of shoulder joint. Ann Surg 132:1052, 1950.

Glousman, RE and Jobe, FW: How to detect and manage the unstable shoulder. J Musculoskeletal Medicine, March, 1990, p 93.

Gurd, FB: The clavicle is an unnecessary bone. Surplus parts of the skeleton: Recommendation for excision of certain portions as a means of shortening the period of disability following trauma. Am J Surg 74:705, 1947.

Jobe, FW, Moynes, DR, and Brewster, CE: Rehabilitation of shoulder joint instabilities. Orthop Clin North Am 18:473, 1987.

Kocher, T: Ein neue reductions metchode fur schulterverrenkung. Berlin Klin 7:101, 1870.

Matson, FA and Zuckerman, JD: Anterior glenohumeral instability. Clin Sports Med 2:319, 1983.

Moseley, HF: Athletic injuries to the shoulder region. Am J Surg 98:401, 1959.

Moseley, HF and Overgaard, B: Hermodsson's Roentgenological Studies of Traumatic and Recurrent Anterior and Inferior Dislocations of the Shoulder Joint. McGill University Press, Montreal, 1963.

Nash, J: Kocher's method of reducing dislocation of the shoulder. J Bone Joint Surg 16:535, 1934.

Neviaser, RJ: Injuries to the clavicle and acromioclavicular joint. Orthop Clin North Am 18:433, 1987.

Oppenheimer, A: Lesions of the acromioclavicular joint causing pain and disability of the shoulder. Am J Roentgenol 51:699, 1944.

Poirier, P and Rieffel, H: Mechanisme des luxations subacromiales de la clavicule, leur traitment par la suture osseuse. Arch Gen Med 1:396, 1891.

Rowe, CR: The Bankhart procedure: Improvements and options. Surg Rounds for Orthopaedics, February, 1990, p 15.

Rowe, CR and Zarins, B: Recurrent transient subluxation of the shoulder. J Bone Joint Surg 63A:863, 1981.

Taft, TN, Wilson, FC, and Oglesby, JW: Dislocation of the acromioclavicular joint: An end-result study. J Bone Joint Surg 69A:1045, 1987.

Thorndike, A and Quigley, TB: Injuries in the acromioclavicular joint. Amer J Surg, 55:250, February, 1942.

Urist, MR: Complete dislocation of acromioclavicular joint. J Bone Joint Surg 28:813, 1946.

CHAPTER 7

Sports Injuries to the Shoulder

Many people of all ages and with varying degrees of physical fitness are active participants in athletics in modern day society; as a result, injuries are becoming more widespread. Sports medicine is now a subspecialty for orthopedic surgeons.

Injuries to the shoulder from sports activities are also being analyzed in kinesthetics, and many new developments are leading to a better knowledge of cause and effects of these sports injuries. Many terms, such as *overuse*, are also being studied by pathologists, kinesiologists, and therapists. Well-documented pathologic conditions such as tendinitis, degenerative cuff disease, and rupture of rotator cuffs are more frequently being found to be the result of sports activities. Treatment and prevention are being highlighted.

It must be understood also that shoulder injuries sustained in athletic injuries in the older population differ from those in younger people participating in the same athletic activity. The kinesthetics of the activity are identical, as are the mechanical stresses placed upon the shoulder, but there is a major difference in the shoulder structures as a result of the aging process.

The older person has preexisting degenerative changes. The overhanging acromial process may be eburnated, causing overgrowth of the anterior margin of the acromium and a thickening of the coracoacromial ligament. There are postural changes in aging that alter the alignment of these structures. The rotator cuff may already have undergone degenerative changes from the narrowing of the suprahumeral space. This narrowing compresses the normally tedious vascular supply to the conjoined tendon (see Fig. 1–24), which predisposes to further degeneration, partial tearing, and even complete tearing.

In the younger athlete there is greater effort and exertion. Longer periods of strenuous athletic activities are more apt to be pursued than in the aged. Microtrauma impairs the stability of the glenohumeral joint, and adequate rest and repair may be overlooked. The microtrauma may elongate the capsular structures, leading to glenohumeral joint instability from degrees of subluxation, and the normal glenohumeral joint motion becomes unstable with greater impingement of the rotator cuff. This status of continuous repetitive changes from numerous microtraumas has been well documented by Jobe and others (1984).

The tissues that normally provide the stability of the glenohumeral joint are the glenohumeral ligaments and their attachment to the glenoid labrum. Especially affected are the inferior glenohumeral ligament (see Fig. 1–14) and the anterior inferior aspect of the glenoid labrum. These can be considered the *static stabilizers.*

The *dynamic stabilizers* are the muscles that apply the force with appropriate leverage during the athletic activity.

Range of motion exercises usually do not relate to the range necessary for a particular sport. Strengthening exercises also are arbitrary rather than specific as to which muscle, through which range, or whether they are for strength or endurance. The manner of doing exercises—for instance, increasing flexibility—is also imprecise. *Full range of motion* is not a clear description in respect to the range needed for a specific sports activity.

When *total body activities* are used in a specific athletic activity, if one merely stretches, for instance, *tight hamstrings* and ignores the flexibility of the shoulder, the result will be failure. In playing tennis, there must be full flexibility of the legs as well as of the shoulders, and in running, full flexibility of the shoulders as well as of the legs. *No athletic activity employs only one extremity.*

In tennis, the serve requires extreme external rotation of the arm, much past 90°. The total body also participates, with the trunk laterally rotated. The serve then becomes a trunk derotation and flexion as the arm descends from an extreme backward externally rotated position (Fig. 7–1).

In the forward descent of the arm with simultaneous derotation (inward) rotation of the shoulder, it is apparent that these muscles must also be strengthened. These are the latissimus dorsi, subscapularis, pectoralis major, and the triceps.

In an overhead pitch, a baseball pitcher similarly uses all the trunk muscles and the hyperextended, posteriorly flexed, externally rotated range of motion, and derotation muscular strength of the upper extremity. Through slow-motion photographic studies, the *throwing mechanism* of the overhead pitch has been documented into basic components, and the tissues involved have been deducted from these studies. By understanding the mechanics and the tissues involved in all the phases of the overhead throw, the abnormal and thus painful disabling results of injury can be determined.

The pitching motion has been divided into five stages (Jobe and associates, 1983, 1984; Tullos and King). Stage 1 is the windup, or a preparatory phase. Stage 2 is early *cocking* (Fig. 7–2), when the ball is in the pitching arm, which goes into abduction, posterior flexion, and external rotation. The weight of the body is on the rear leg, and the trunk is rotated away from the batter. Stage 3 is termed *late cocking* (Fig. 7–3), in which the entire body is shifted to the forward foot, the trunk begins derotation, and the arm is now in *maximum* external ro-

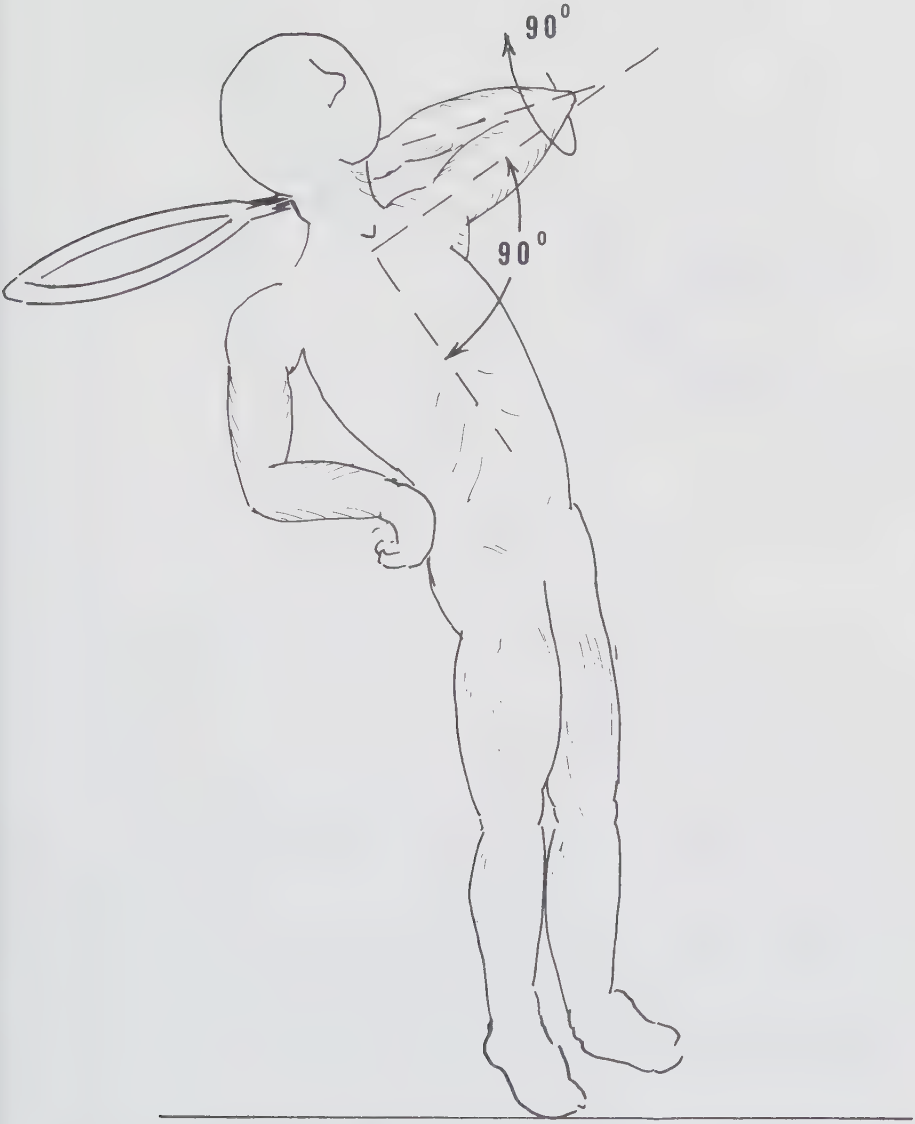


Figure 7–1. Total derotation of trunk and arm during a tennis serve. In the tennis serve the arm at abduction demands extreme external rotation to achieve power. The trunk then couples the forces of derotation. Each degree of external rotation has been equated with miles per hour of the serve.

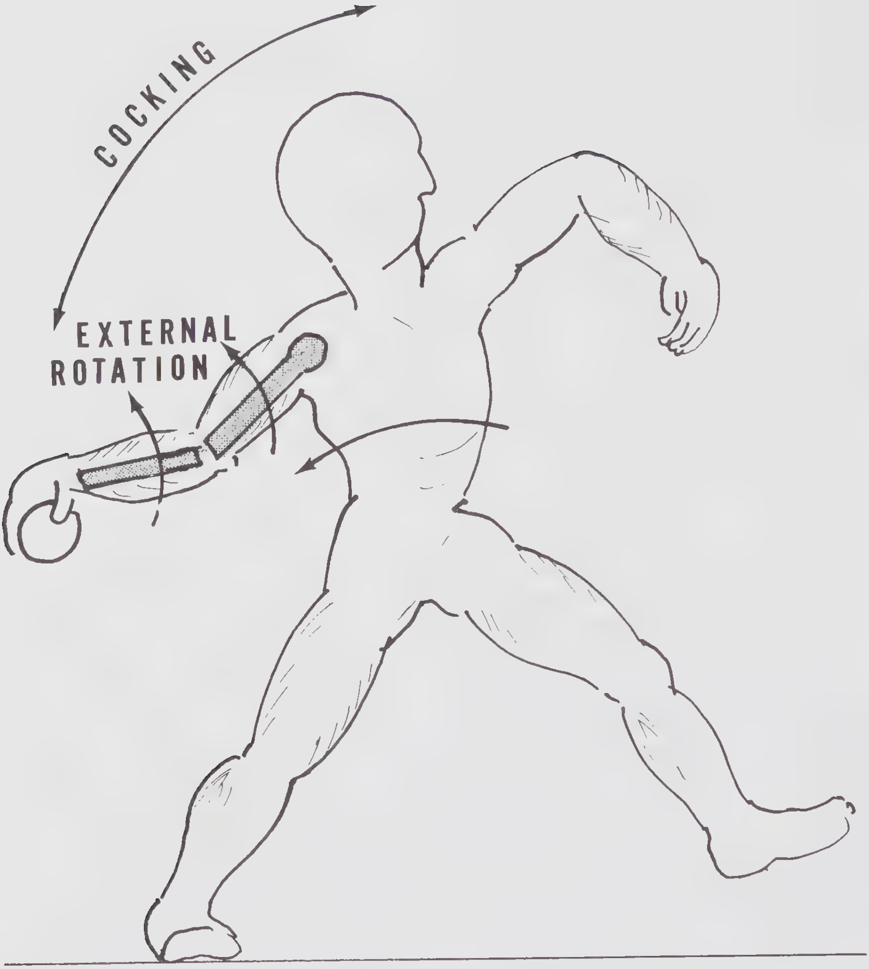


Figure 7–2. Cocking—second stage of throwing mechanism. After the windup, when the trunk rotates to the right and the ball is placed in the throwing hand, the second stage known as *cocking* places the arm in maximum external rotation and posterior flexion. The deltoid muscle is very active in this stage and appears before the rotator cuff becomes engaged. Compression of the rotator cuff occurs during this stage.

tation and posterior flexion. With these motions there is great exposure of the rotator cuff tendon as it passes under the overhanging acromium and coracoacromial ligament. In the pitching stages, the deltoid becomes very active in forceful abduction. This muscle, by its anatomic alignment, causes upward displacement of the humerus, causing impingement of the cuff tendon against the overhanging structures.

In stage 2 of the throwing mechanism, the *cocking* stage, in which the arm

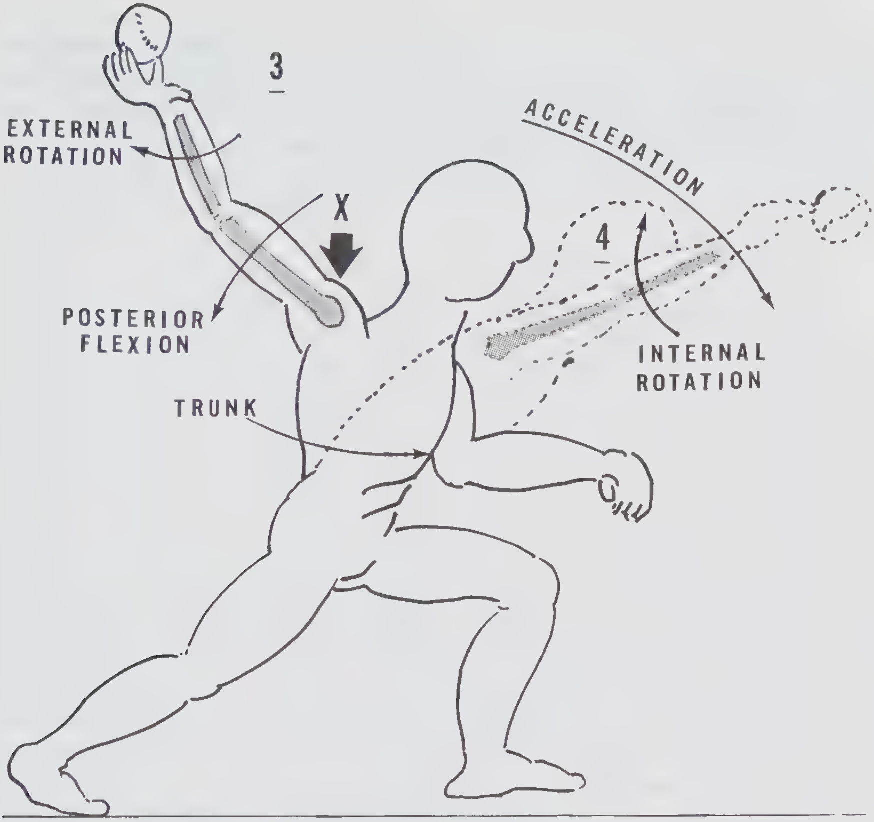


Figure 7–3. Overhead pitching motion: stages 3 and 4.

The pitching motion has been divided into five stages: (1) windup; (2) early cocking; (3) late cocking (X), in which the arm achieves extreme external rotation and hyperextension; (4) acceleration, in which internal rotation precedes release of ball; and (5) follow-through stage.

In stage 3 the head of the humerus is against the inferior anterior capsule with extreme external rotation. At this stage the capsule or the labrum can be torn. Because the head of the humerus is also deep under the arch, there is maximum entrapment of the cuff.

In stage 4 the arm accelerates, placing traction stress upon all the musculotendinous tissues from the triceps, subscapularis, and latissimus dorsi muscles especially.

is posterior to the body at the onset of elevation, abduction, and external rotation until the arm is overhead, there is early deltoid contraction ahead of supraspinatus contraction. This is the stage in which there is greater cuff tendon impingement. If there is tendon degeneration or supraspinatus muscle weakness, the deltoid becomes even more overwhelming and further degeneration results.

During stage 3 (see Fig. 7–3), the acceleration stage, there is minimal rotator cuff activity but great pectoralis major and latissimus dorsi action inasmuch as these are powerful internal rotators and downward flexors. In the follow-through stage (stages 4 to 5), when the arm moves downward and across the lower body, the rotator cuff is undergoing *eccentric* (deceleration) contraction, which is the most stressful to the rotator cuff. This can be a *traction* injury upon the cuff.

When the deltoid muscle acts unopposed, as from a weakened or damaged cuff, greater impingement results. Fatigue of the scapulothoracic muscles also enhances the impingement.

Diagnosis requires evaluating every muscle involved in the scapulohumeral action and determining at which aspect of shoulder girdle function pain occurs. X-ray studies, including arthrography (more currently MRI), are diagnostic, but arthroscopic examination is proving to be even more fruitful. Kinetic x-ray films combined with simultaneously invoked electromyography (EMG) are the studies of the future to determine at which phase in any athletic activity there is impairment, impingement, and even muscular weakness. Athletic ergonomics will be a study of promise in the future.

Stage 4 is termed the *acceleration stage* because the arm now begins retrotation into internal rotation and forward flexion down and across the chest. The body weight is now on the forward foot, and the trunk is significantly rerotated. The ball is released. Stage 5 is the *follow-through stage*, or the deceleration stage.

Remembering the neuro-orthopedic control of precise actions of extremities, it is apparent that proper conditioning and—even more important—proper *body mechanics* of the total arm *and* the trunk must be employed. Even with complete normalcy of all the tissues of the shoulder complex any deviation from normal action can traumatize the involved tissues.

In stages 3 and 4, with the arm in maximum external rotation and posteriorly flexed, the head of the humerus is contained exclusively by the anteroinferior capsule. The rotator cuff is anatomically unable to prevent subluxation. It is at this stage that the capsule or glenoid fossa labrum can be torn.

The arm is severely abducted and posteriorly flexed, and the greater tuberosity with its rotator cuff and the biceps tendon is in forced contact with the overhanging acromium and coracoacromial ligament. The downward rotators and internal rotators exert a powerful force on the humerus, with simultaneous upward motion of the humeral head. *Entrapment* of the cuff tendon and biceps tendon can occur. Rotator cuff tendinitis and bicep tendinitis are the possible sequelae.

As the arm decelerates (stages 4 to 5), a powerful stress is imposed on the posterior structures, including the triceps and latissimus dorsi. There is force against the posterior capsule during this stage.

When a pitcher or tennis player, who has been subjected to these forces, presents with a painful shoulder, he or she presents with *global shoulder pain*. Only precise examination can determine which of the involved shoulder tissues is the culprit.

Because such a complex neuromuscular mechanism is involved in pitching in baseball and serving in tennis (and other similar athletic endeavors), repeated efforts can cause accumulative effect. Minor insults with tissue changes may recur without sufficient time for repair. The tissues, especially tendons and capsules, subjected to extreme stretch may become attenuated. Muscles can be subjected to *overuse*, pathologic changes. Warm-up strengthening and flexibility exercises and avoidance of fatigue are mandatory.

Diagnosis of the specific impaired tissue is important. Inspection of the shoulder girdle reveals atrophy, specific muscular weakness, impaired range of motion, crepitation, and local tenderness. Because most of these tests have been discussed they will not be repeated here. Suffice it to say they include observation of abduction, external rotation, and overhead elevation; active and passive range of motion; strength of the abductors and external rotators; atrophy; and local tenderness. Each of these observations and tests specifies whether the pathologic process is in the capsule, ligament, or muscles.

Several specific tests for *sports shoulder injury* need amplification. Subluxation of the glenohumeral joints with capsular or glenoid labrum tears must be ascertained. From the hyperextension, excessive external rotation, and overhead elevation in many athletic activities the anteroinferior aspect of the capsule and the anteroinferior aspect of the glenoid labrum are subjected to tearing.

These tests for subluxation are termed *apprehension and relocation tests.* The apprehension test (Fig. 7–4) is performed with the patient in the supine position with the arm abducted to 90° and externally rotated. The examiner pulls the head of the humerus in an anterior position while holding the elbow fixed. Patients who have had recurrent subluxations will express apprehension but not necessarily pain. Patients who have an anterior subluxation experience pain but not apprehension. There is greater joint *play* (motion) in the involved shoulder than in the compared normal contralateral side.

The integrity of the posterior capsule and labrum is similarly tested but in the opposite direction. With the same patient position, the arm is posteriorly moved at the glenohumeral joint (see Fig. 7–3), and either apprehension is expressed or there is pain in posterior motion of the glenohumeral joint. This posterior capsular test is termed *relocation testing.*

Often a subluxation test may be questionable with the patient awake and must be performed under an anesthetic when subluxation can be clearly demonstrated.

Routine radiologic tests (x-ray studies) are of limited value in glenohumeral subluxation, but CT, MRI, arthrography, and even arthroscopic examinations are usually diagnostic. Whether treatment is to be surgical or nonsurgical will then be determined (Jobe and Kvitne).

The range of motion of the shoulder can be elicited in the following manner (Fig. 7–5): In the supine position with the arm at the edge of the bed (table), at 90° to the trunk and the elbow bent to 90°, the patient must externally rotate the arm. A weight can be used to increase greater flexibility. A slow, repeated *stretching* of the internal rotators will ensue, but this increase in range of motion requires many weeks of repeated stretching exercise, especially preceding the athletic activity.

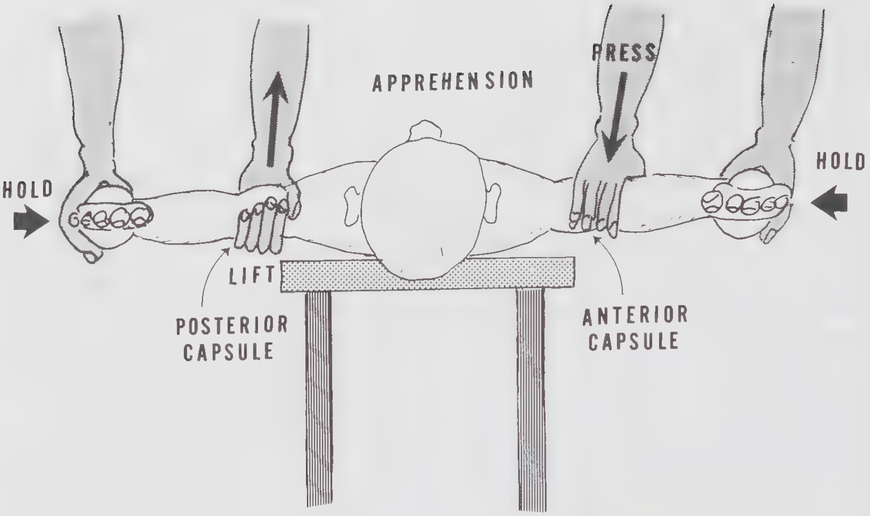


Figure 7–4. Apprehension and relocation tests for shoulder subluxation. With the patient supine, the arm at horizontal abduction, the elbow flexed 90°, and the upper arm externally rotated, the tests are performed: Anterior capsule (*right arm in illustration*), the elbow is supported and there is downward pressure upon the upper arm. In recurrent subluxations there is *apprehension* by the patient. In an existing subluxation excessive motion and pain are elicited.

To test the posterior capsule *(relocation) (left arm in illustration)* the elbow is supported and the upper arm is elevated. In patients with previous subluxations, apprehension is noted. In present subluxation, there is excessive motion and pain.

In bowling, for instance, the arm must hyperextend posteriorly with simultaneous rotation (Fig. 7–6). In the *swing through* forward flexion that follows, there is also rotation, which has been begun at the start of the backward posterior flexion.

Total flexibility exercises for the shoulder posterior flexion are depicted in Figure 7–7, using a piece of standard furniture. Figure 7–8 (Dips) exemplifies the hyperextension of the shoulder with strengthening when done actively against body weight resistance.

Of interest, a recent report (unpublished) by Prichard, director of Somax Posture and Sports in Corte Madera, California (New York Times of April 16, 1990), in which long distance (marathon) running was evaluated revealed that limited posterior shoulder flexibility increased the running distance of the athlete.

This apparent paradox is based on the angle of the legs during running and the legs' relationship to their center of gravity. As depicted in Figure 7–9, this angle can be drawn from envisioning the runner anteriorly and drawing this angle. If the angle is 3°, the distance run is shorter than if the angle exceeds 10°. In the shorter angle there are shorter steps as the non-weight-bearing leg has

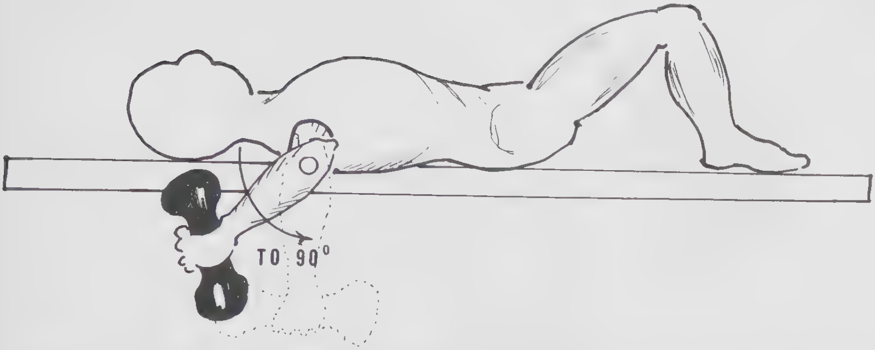


Figure 7–5. Stretch exercise for increased external rotation. In the supine position with the arm past the edge of the bed (table) and the arm at 90° abduction, a weight in the hand increases external rotation toward 90°.

to *cross over* a longer distance. With an angle of 10°, due to lateral motion of the pelvis, the non-weight-bearing foot has to cross over further, and thus the runner takes longer steps during the stride. The distance run is the same, but the distance of the steps taken is greater. A runner takes approximately 1000 strides per mile. If each step has to go a greater distance—even if only several inches—the distance run is longer when totalled.

The difference of going from 3° to 10° has been ascertained to be that in the former (3°) the shoulder swings posteriorly further, whereas in the latter (10°) there is less posterior flexion, which is *made up* by accentuating the vertical motion, rotation, and lateral swing of the pelvis, hence there is more distance of leg crossover.

As also stated, entire body flexibility is needed rather than stretching only the extremity involved. A *swinging warm-up exercise* is depicted in Figure 7–10 as an example.

Trunk twists occur in almost all upper extremity sports activities, thus the trunk needs flexibility in rotation (Fig. 7–11). Lateral trunk flexion is a separate flexibility exercise to complement the trunk rotation (Figs. 7–12, 7–13).

Total body flexibility is emphasized rather than only the involved extremity because if there is limitation of a portion of the trunk, extra—and often excessive—demand of range of motion is placed upon the upper extremity.

In most sports there is great demand upon shoulder rotation, posterior flexion, and overhead extension.

TREATMENT OF THE INJURED ATHLETE

Treatment of the injured athlete demands an approach slightly different from that of a nonathlete. Immobilization and inactivity are the enemies of ath-

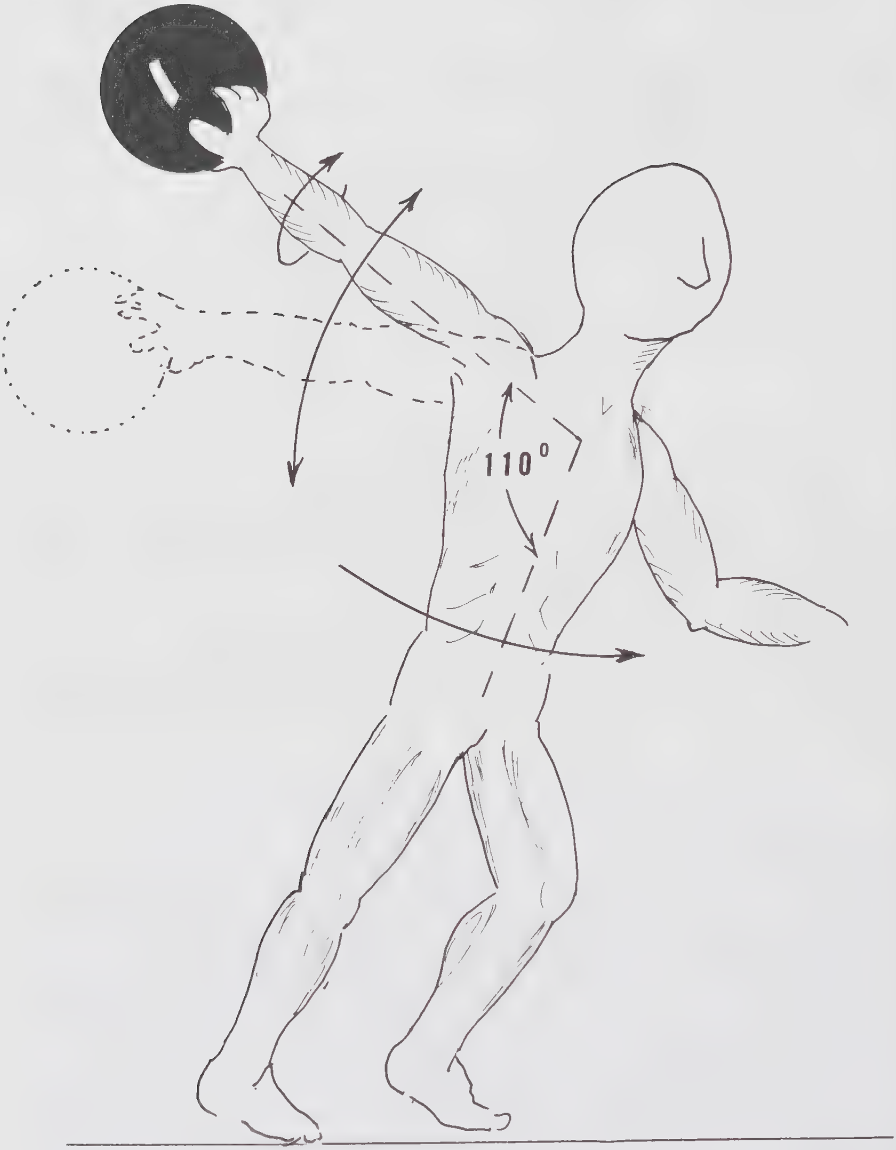


Figure 7–6. Hyperextension needed for better bowling. In the swing phase of bowling, the arm must hyperextend while rotating. This range of motion must be gained during warm-up exercises before bowling.

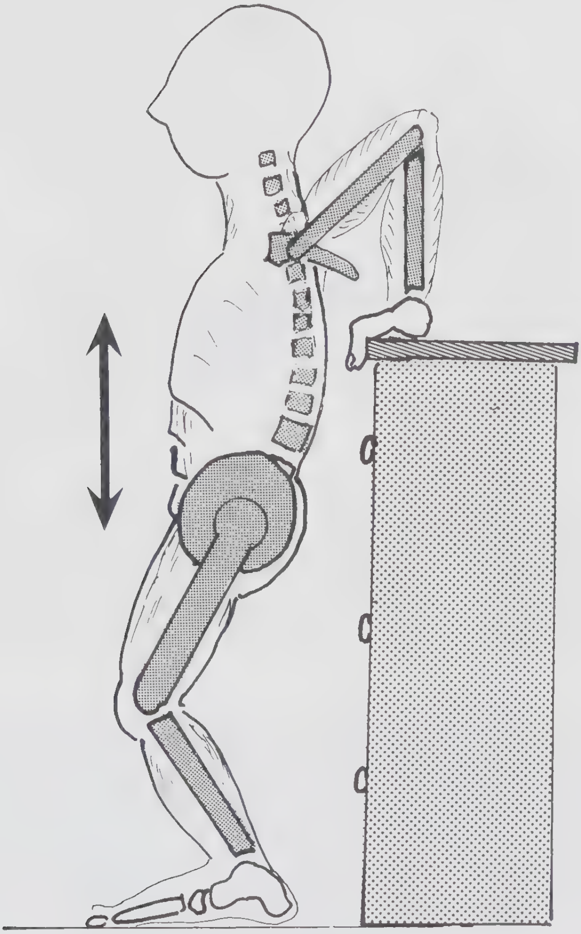


Figure 7–7. Hyperextension exercise of the shoulder. By merely doing deep knee bends of varying degrees, with the arm at different levels while holding the full flexed-knee position for varying periods, the anterior capsule of the shoulder can become more flexible. (From Cailliet, R and Gross, L, with permission.)

letes. These two provoke atrophy and deconditioning, which can lead to a failure to resume athletic prowess to the preinjury status.

Acute injury is indicated by the cardinals of inflammation: pain, warmth, erythema, swelling, and—more significantly—loss of function. A defense reflex mechanism occurs from pain and fear after injury, which causes nonuse of the extremity (Smodlaka). This vicious cycle must be broken early if complete recovery is to be attained.

Early intervention of the pain of acute injury can be initiated by injection

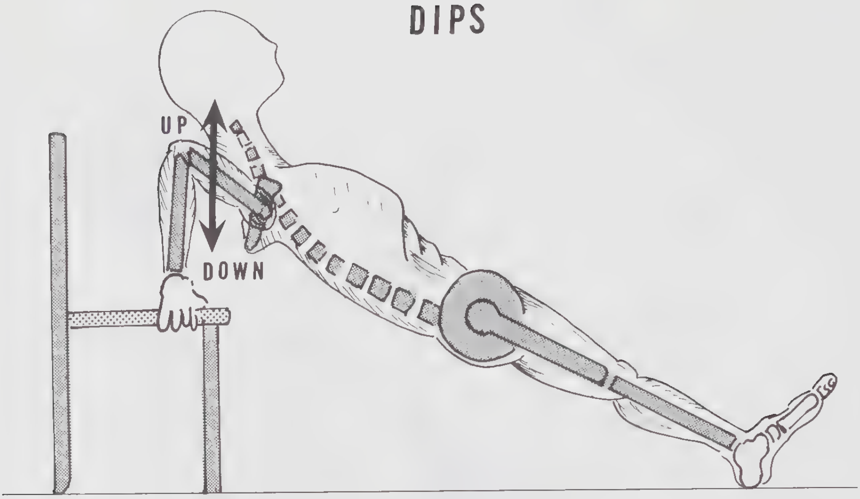


Figure 7–8. Dips exercises for hyperextension of the shoulder posteriorly. With the body supine and hands on a chair, the weight of the body causes an increase in shoulder range of motion. The motion of a push-up from this position strengthens the muscles of the shoulder. (From Cailliet, R and Gross, L, with permission.)

of a local anesthetic. This breaks up the cycle but is fraught with danger. Because most athletes wish to return immediately to sports, they may have false security and, now with *no* pain, there is the erroneous implication that there is no longer any damage to the involved tissues. The attitude that rest of the part is not only no longer necessary but also detrimental or that one must work it out—no pain, no gain—may be the credo most detrimental to the patient.

The pain and edema after acute injury are caused by formation of nociceptors and microscopic extravasation of blood, lymph, and even synovial fluids within the joint. This extravasation and lymph formation around a joint or tendon may result in chronic organized edema, fibrosis, and adhesions. These sequelae have been recognized as early as the fourth day after injury (Kottke).

In a Robert Jones lecture (Perkins) it was stated, "It is difficult to say when the inflammation has ceased and repair has begun. It is therefore difficult clinically to determine when to alter the proposed treatment."

The general principles of acute therapy can be applied to the shoulder, with minor modifications:

1. Immobilize the affected part. In the shoulder this is difficult because splinting and avoidance of use in activities of daily living are barely possible. Any activity not needed for general daily activities must be discouraged. Avoidance of athletic activities is mandatory.

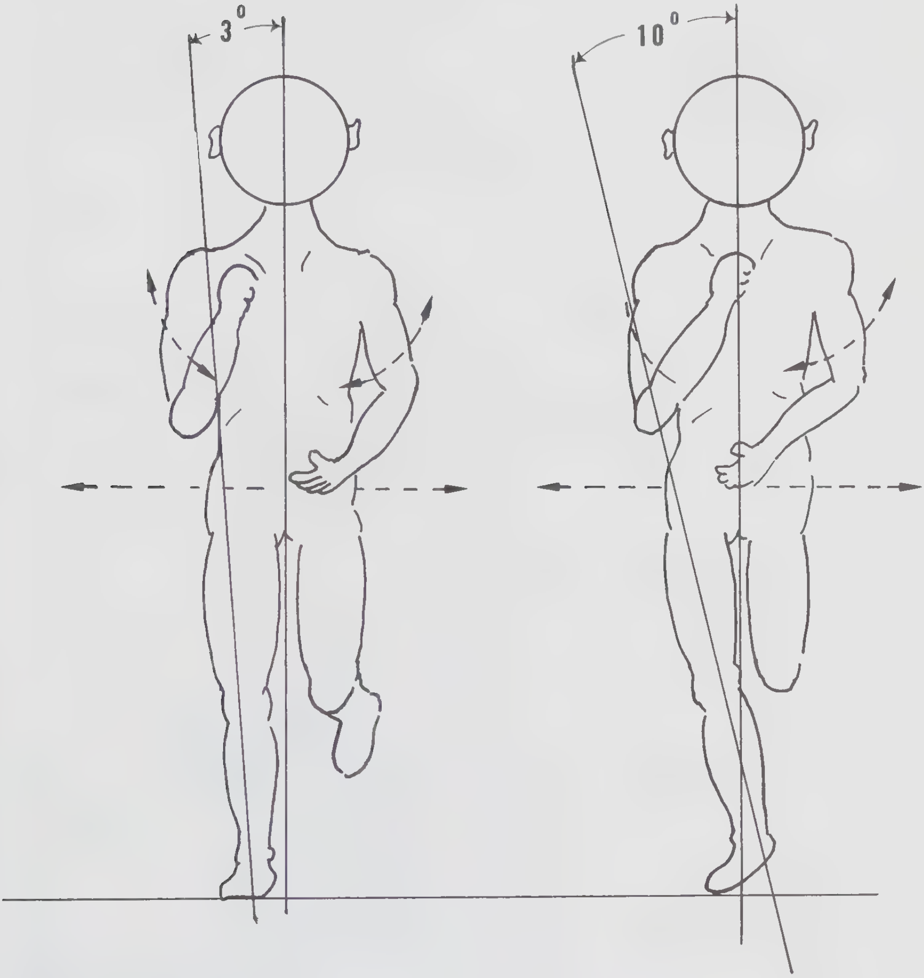


Figure 7–9. Leg gravity angle during running. At 3° angulation the stride is effective due to shoulder posterior-anterior swing. At 10° angulation the stride is longer, due to crossover.

2. Elevation of the injured part above the heart level to decrease edema is appropriate, but in the shoulder this may initiate pain. Common sense prevails.
3. Ice applied locally three to five times daily over 72 hours is valuable to decrease the pain, to reduce spasm, to prevent formation of nociceptors—especially the histaminelike products—and to decrease the formation of further microscopic bleeding.

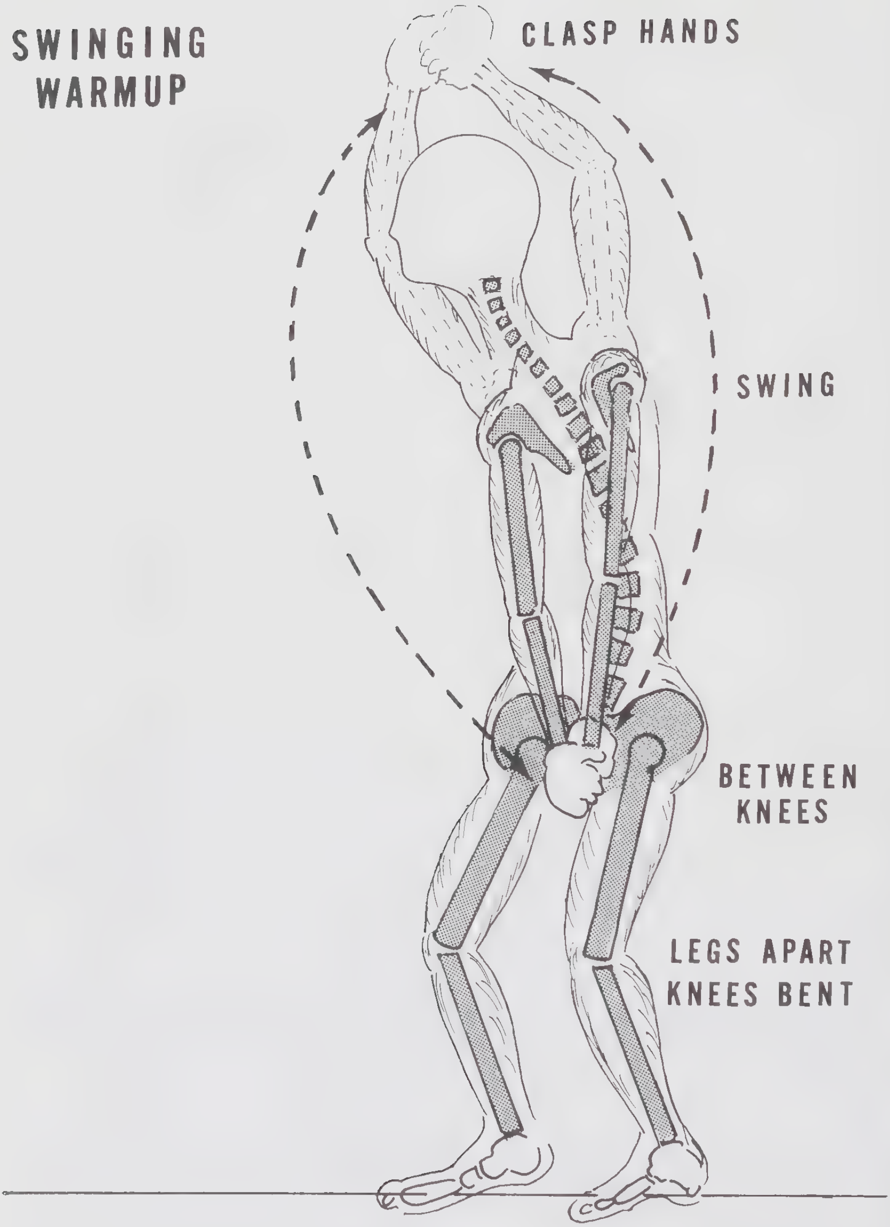

SWINGING
WARMUP
CLASP HANDS
SWING
BETWEEN
KNEES
LEGS APART
KNEES BENT

Figure 7–10. Swinging warm-up rotatory trunk flexibility exercise. With the legs slightly apart and the arms overhead, hands clenched, the arms are brought diagonally down and across to the opposite knee. Smooth repeated exercise done on both sides is a good warm-up exercise for any sport. (From Cailliet, R and Gross, L, with permission.)

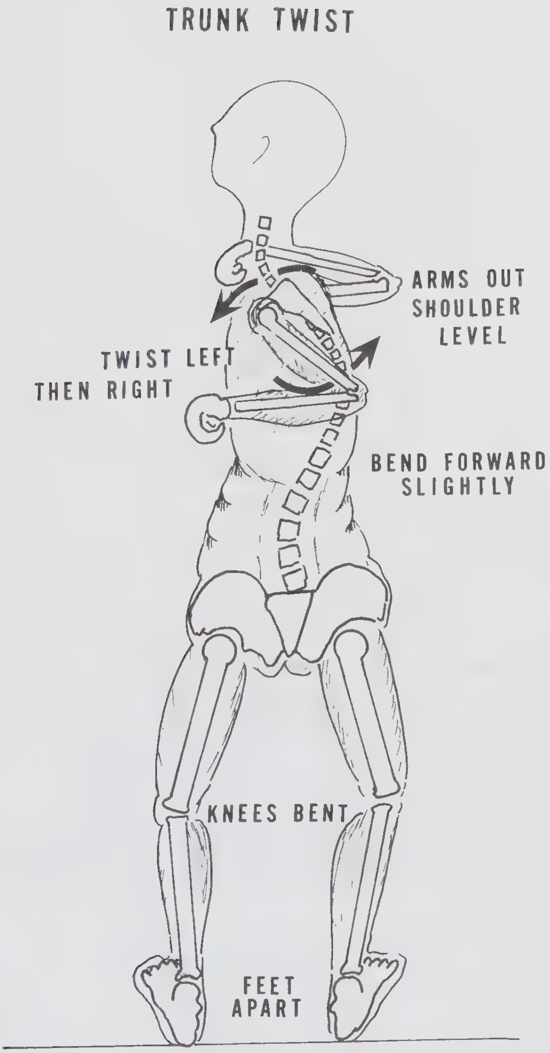

TRUNK TWIST
ARMS OUT
SHOULDER
LEVEL
TWIST LEFT
THEN RIGHT
BEND FORWARD
SLIGHTLY
KNEES BENT
FEET
APART

Figure 7–11. Trunk twist exercise. With the feet slightly apart and the knees slightly bent, the arms flexed at the elbows and abducted at the shoulder, the trunk is rotated as far as possible to the left, then the right, slowly and smoothly, to increase each rotation to tolerance. (From Cailliet, R and Gross, L, with permission.)

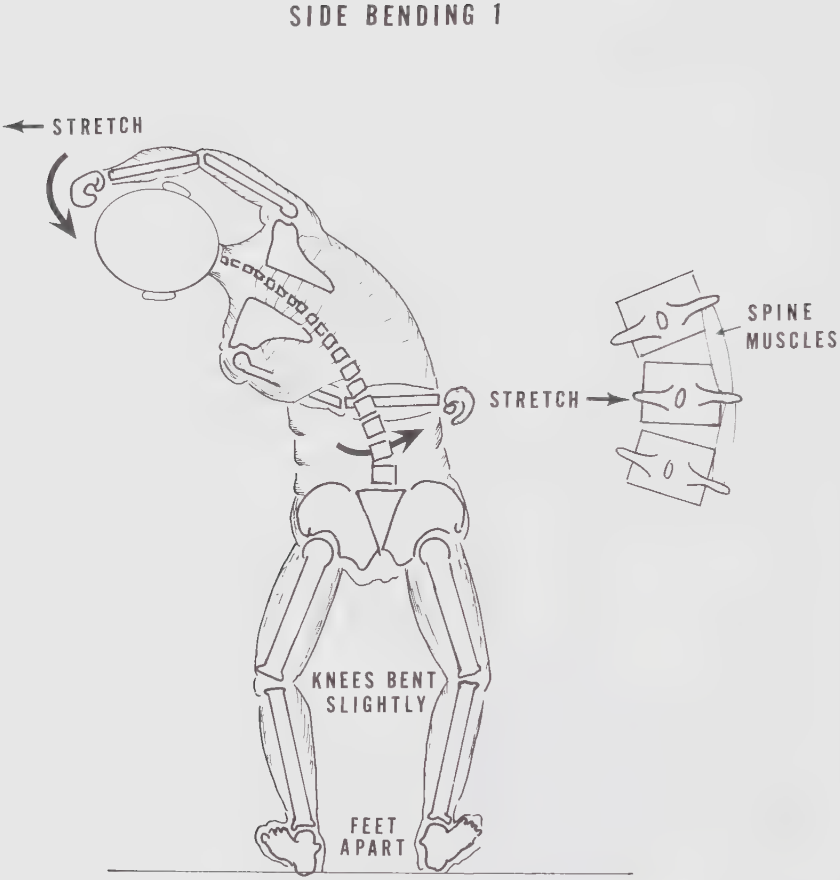


Figure 7–12. Side bending 1. By slow undulating lateral trunk flexion first to the left, then the right, the lateral trunk flexibility increases. The overhead arm also increases shoulder—scapular and glenohumeral—range of motion. (From Cailliet, R and Gross, L, with permission.)

4. Compression, so valuable in most extremity injuries, is difficult in the shoulder.
5. Aspirate any evident effusion or hematoma.
6. Early gentle passive then active range of motion exercises are recommended, avoiding the extremes of range.
7. Isometric muscular contraction exercises do not mobilize the joint but cause dispersal of accumulated fluids and maintain muscle tone.
8. With good judgment, rehabilitation exercises to strengthen the affected muscles must be initiated early. In the shoulder this relates especially to the subscapularis external rotators and the deltoid, pectoralis major,

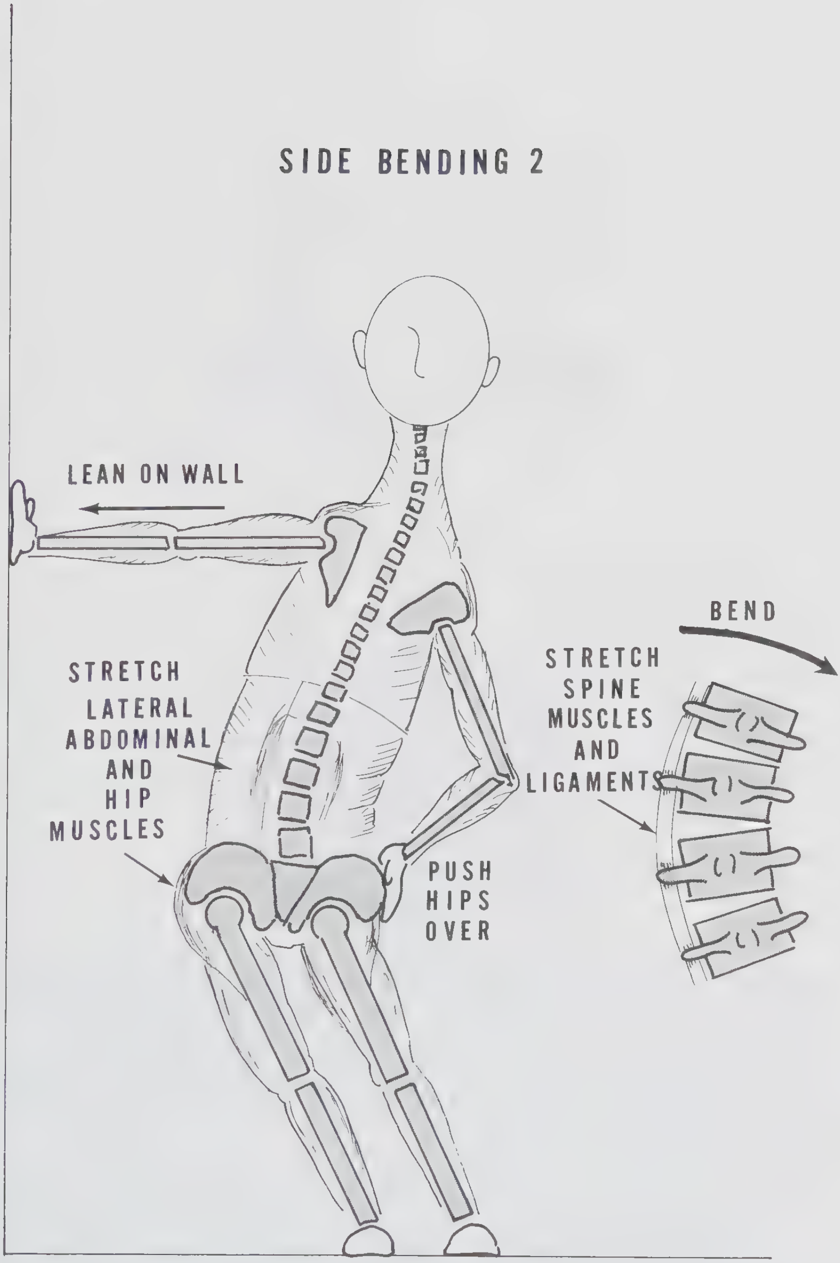

SIDE BENDING 2
LEAN ON WALL
BEND
STRETCH LATERAL ABDOMINAL AND HIP MUSCLES
STRETCH SPINE MUSCLES AND LIGAMENTS
PUSH HIPS OVER

Figure 7–13. Side bending 2. By extending the arm horizontally to support the body away from the wall, the trunk becomes stretched. Lateral flexibility is increased by the opposite hand pressing the pelvis toward the wall. (From Cailliet, R and Gross, L, with permission.)

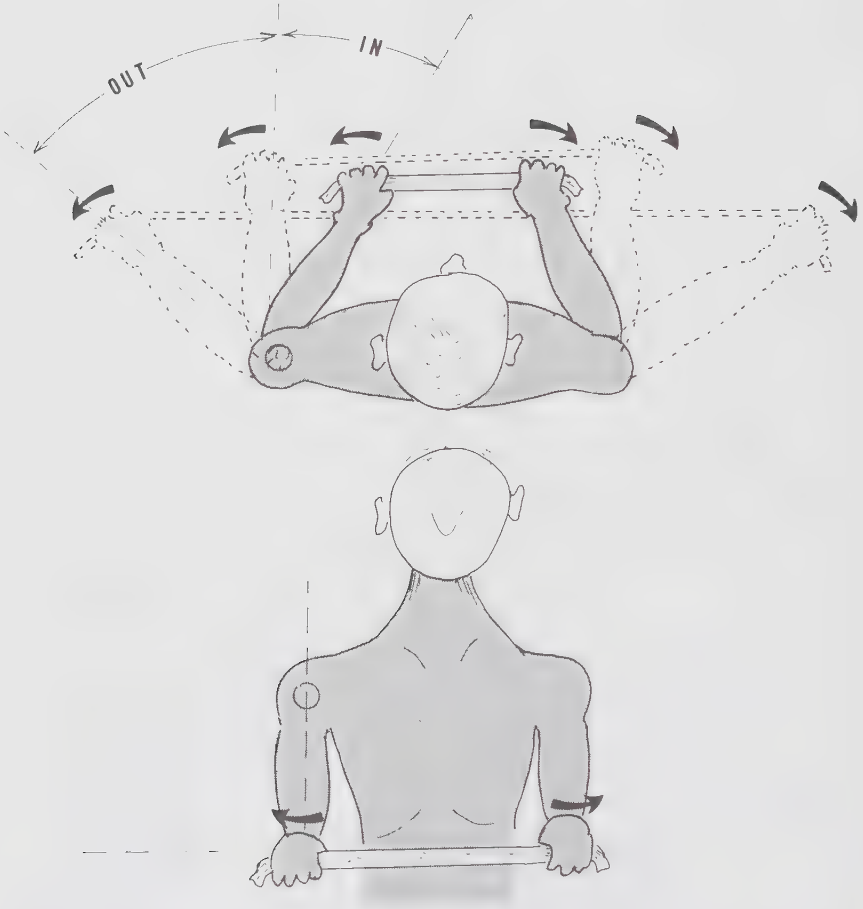


Figure 7–14. Strengthening exercises for the rotator cuff. With the elbows at the side and flexed 90°, an elastic of varying resilience and length is used to resist external rotation. With the hands almost together, external rotation is performed within that range of motion. With the hands in the sagittal plane, a greater degree of external rotation is exercised. Ultimately the arms are fully rotated externally. Varying degrees of resiliency of elastic may be used or *spring stretchers.*

and the long head of the biceps (Noah and Ramesh). Because the rotator cuff muscle and its tendon are the most frequently damaged and require major rehabilitation, these exercises will be emphasized. Slow gradually progressive exercises strengthen not only the muscle but also its tendon. They must be initiated early, progressively, and they must be continued indefinitely.

External Rotator Exercise

Strengthening exercises of the rotator cuff, especially of the supraspinatus muscle tendon, are very important to maintain the health of the soft tissues of the cuff. This exercise is depicted in Figure 7–14. The arm must be kept with the elbow at the side, flexed to 90°, and externally rotated about this axis. With the hands approximated in front of the body, the range of external rotation is in that range. As the elastic, used for resistance, is elongated, the range of external rotation increases. *The elbow must be kept at the side* to avoid abduction of the humerus at the glenohumeral joint.

The long head of the biceps, which keeps the head of the humerus depressed during abduction, may be strengthened by forward flexion exercises, against resistance, but the forward flexion must be limited to 90° flexion. The pectoralis major muscle and the anterior deltoid muscle must also be strengthened by horizontal adduction resisted exercises with the arm abducted at 90° and also with the arm at the side.

When there are significantly damaged tissues that will not or do not respond to conservative nonsurgical treatment, surgical intervention may be sought. The surgical procedures are beyond the scope of this chapter, but they include acromioplasty, subacromial decompression, partial severance of the coracoacromial ligament, repair of the rotator tendon, and others through an arthroscopic approach or open surgery. The decision will depend on the expertise of the consulting orthopedic surgeon.

Presurgical and postsurgical physical therapy will ensure better recovery regardless of the efficacy of the surgical intervention.

BIBLIOGRAPHY

Cailliet, R and Gross, L: The Rejuvenation Strategy. Doubleday & Co., New York, 1987.

Jobe, FW, et al: An EMG analysis of the shoulder in throwing and pitching: A preliminary report. Am J Sports Med 2 (1):3, 1983.

Jobe, FW, et al: An EMG analysis of the shoulder in pitching: A second report. Am J Sports Med 12 (2):218, 1984.

Jobe, FW and Kvitne, RS: Shoulder pain in the overhand and rotator cuff impingement. Orthop Review 18(9): 1989.

Kottke, FJ: Therapeutic Exercises. In Krusen, F, et al: Handbook of Physical Medicine and Rehabilitation, ed 2. WB Saunders, Philadelphia, 1971, pp. 385.

Noah, J and Ramesh, G: Rotator cuff injuries in the throwing athlete. Orthopaedic Review 17(11):1091, 1988.

Perkins, G: Rest and Movement. J Bone Joint Surg (B) 35:521, 1953.

Smodlaka, V: Rehabilitating the Injured Athlete. The Physician and Sports Medicine, Vol 5, no 11. McGraw-Hill, 1977.

Tullos, HS and King, JW: Throwing Mechanism in Sports. Orthop Clin North Am 4 (3):709, 1973.

Turkel, SJ, et al: Stabilizing Mechanism Preventing Anterior Dislocation of the Glenohumeral Joint. J Bone Joint Surg 63A:1208, 1981.

CHAPTER 8

Neurologically Referred Shoulder Pain

Pain felt in the shoulder region may be experienced when the site is distal and is neurologically referred to the shoulder region. In most articles concerned with shoulder pain a differential diagnostic statement is made to *rule out cervical referred pain* as the cause of the shoulder pain. All the cervical roots refer to the upper extremity, whether they are at the cervical cord, cervical foramina, brachial plexus, or along the nerve roots in their passage down the upper extremity.

CERVICAL NERVE ROOT INVOLVEMENT (RADICULOPATHY)

Irritation of a nerve root at the cervical level may occur from

1. Mechanical root entrapment at the foraminal level
2. Posttraumatic cervical subluxation
3. Cervical disk herniation
4. Cervical spondylosis (foraminal stenosis)
5. Extracervical entrapment in the brachial plexus and/or peripheral nerves

A typical nerve root (Fig. 8–1), which originates at the cord level, emerges from the cervical spinal canal as fila that merge to form a root, which in turn emerges laterally through the cervical foramen (Fig. 8–2). After emergence from the foramen the nerve proceeds laterally and downward in the cervical

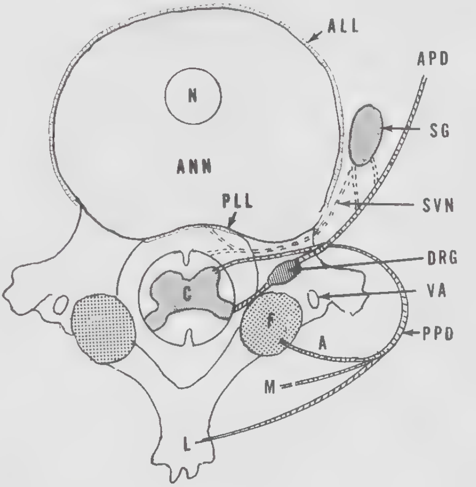


Figure 8–1. Component fibers of a cervical nerve. A view of a functional unit depicts the components of a cervical nerve.

C = spinal cord
SG = stellate ganglion
DRG = dorsal root ganglion
APD = anterior primary division
PPD = posterior primary division
A = articular branch of PPD
M = muscular branch of PPD
L = ligamentous branch of PPD
SVN = sinu vertebral nerve; branches to dura and PPL
N = disk nucleus
ANN = disk annulus
ALL = anterior longitudinal ligament
PLL = posterior longitudinal ligament
F = facet
VA = vertebral artery foramen

grooves (Fig. 8–3) to ultimately form the brachial plexus (Fig. 8–4).

Each nerve root contains sensory, motor, and sympathetic fibers that innervate the upper extremities. The sensory nerve components that supply sensation to the skin area of the upper extremity are termed *dermatomes.* Each nerve has motor roots that innervate the muscles of the upper extremity, termed *myotomes.* It has been estimated that some 30 percent of the motor roots also transmit sensation toward the cord. The sympathetic fibers innervate the blood ves-

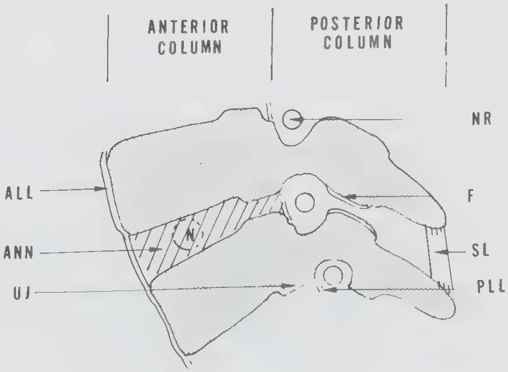


Figure 8–2. Lateral view of a functional unit of the cervical spine C-3 to C-7. The spine is divided into anterior and posterior columns. The component parts are ALL=anterior longitudinal ligament, ANN=annulus fibrosus, UJ=uncovertebral joint, NR=nerve root, F=facet, SL=superior ligament, PLL=posterior longitudinal ligament.

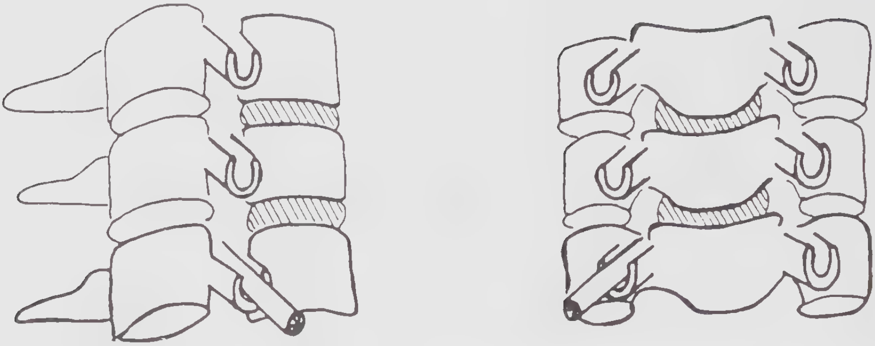


Figure 8–3. Direction of foraminal grooves: diagram depicting the downward-forward direction of the cervical nerve root.

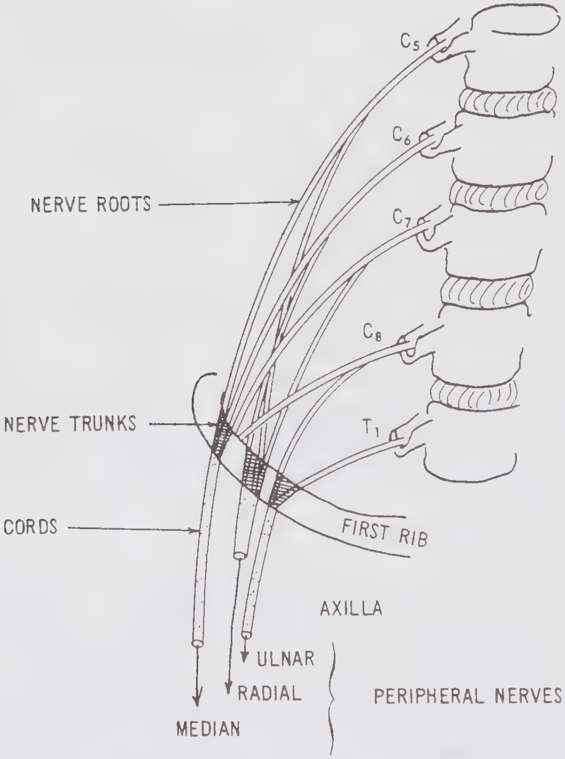

C_5
C_6
C_7
C_8
T_1
NERVE ROOTS
NERVE TRUNKS
CORDS
FIRST RIB
AXILLA
ULNAR
RADIAL
MEDIAN
PERIPHERAL NERVES

Figure 8–4. Brachial plexus (schematic). The brachial plexus is composed of the anterior primary rami of segments C_5, C_6, C_7, C_8, and T_1. The *roots* emerge from the intervertebral foramina through the *scalene muscles.* The roots merge into the *trunks* in the region of the first rib. The trunks via divisions become *cords* that divide into the peripheral nerves of the upper extremities.

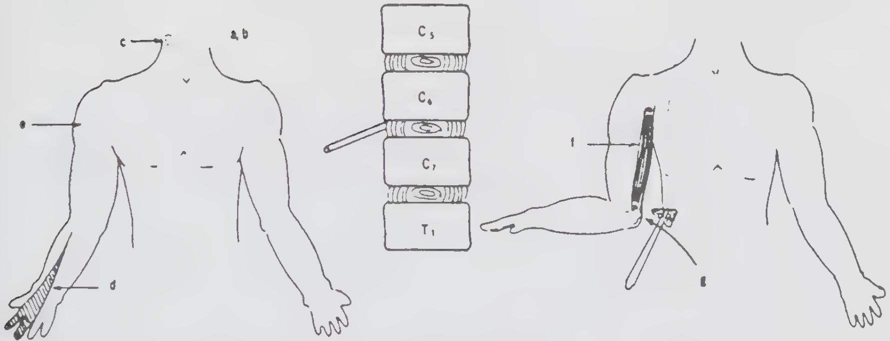
sels, sweat glands, and hair follicles but also transmit sensations termed *paresthesias.*

With an intact unimpaired nerve root the upper extremity has normal sensation and motor function of all the myotomes and dermatomes. With entrapment, compression, or inflammation each nerve root can transmit a painful sensation, an impaired sensation (hypalgesia, hypesthesia, or paresthesia) and/or weakness or paresis of a myotome (Figs. 8–5, 8–6, 8–7).

A summary of referred pain to the upper extremity from cervical radiculitis is depicted in Figure 8–8.

To differentiate shoulder pain from a pathologic process with cervical origin the following criteria must be met:

1. Pain in the upper extremity is initiated by cervical motion, not merely from shoulder movement.
2. Referral of pain is more dermatomal than diffuse in the shoulder girdle.
3. There are usually dermatomal symptoms, such as numbness, tingling, or even pain. These dermatomal symptoms relate to (that is, they can



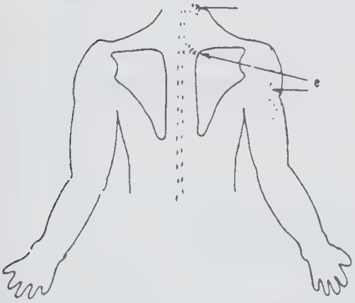


Figure 8–5. Sixth cervical nerve root irritation.

a = Neck rigidity. Limited extension and rotation to the right.

b = Pain and paresthesia aggravated by coughing and sneezing.

c = Tenderness over exit of C_6 nerve root.

d = Paresthesia and hypesthesia of thumb and some of index finger (from history and physical examination).

e = Subjective pain and tenderness over deltoid and rhomboid muscle areas.

f = Weakness of deltoid and biceps muscles.

g = Depressed biceps jerk.

h = x-ray studies equivocal.

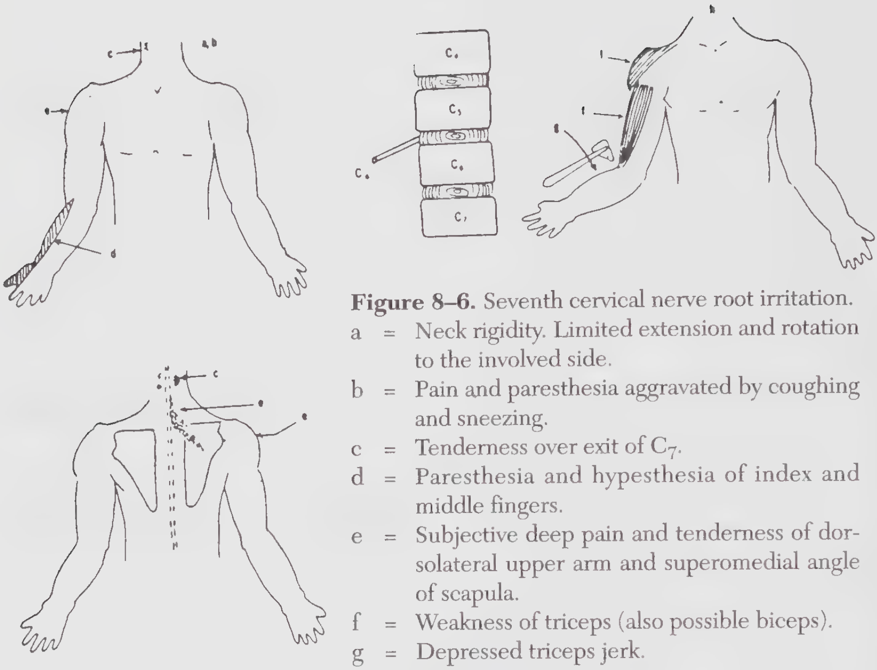


Figure 8–6. Seventh cervical nerve root irritation.

a = Neck rigidity. Limited extension and rotation to the involved side.
b = Pain and paresthesia aggravated by coughing and sneezing.
c = Tenderness over exit of C_7.
d = Paresthesia and hypesthesia of index and middle fingers.
e = Subjective deep pain and tenderness of dorsolateral upper arm and superomedial angle of scapula.
f = Weakness of triceps (also possible biceps).
g = Depressed triceps jerk.

be reproduced by) neck position and/or movement.

4. Dermatomal/myotomal symptoms are usually related to abnormal neurologic signs at the time of examination, that is, specific muscle group weakness, abnormal reflex changes, sensory hypalgesia of a dermatome (Fig. 8–9).
5. There is failure to reproduce symptoms specifically by shoulder motions of abduction, external rotation, painful arc.

It must be remembered that symptoms and signs of a cervical radicular lesion may be present in shoulder girdle lesion, and it requires an astute diagnostician to determine the importance as well as the presence of either. Laboratory confirmatory findings such as abnormal cervical spine x-ray studies must be carefully assigned to the clinical story and findings on examination because they may be totally unrelated.

MECHANISM OF CERVICAL RADICULAR PAIN IN THE UPPER EXTREMITY

Normally the cervical nerve roots emerge through the foramen enclosed in a dural sheath. The nerve roots remain unscathed by neck motion although

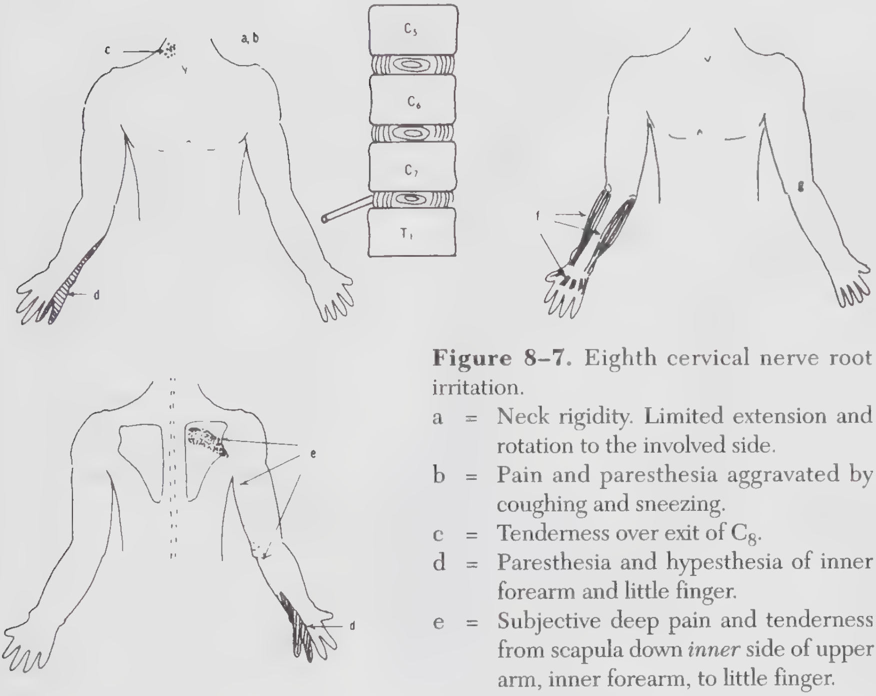


Figure 8–7. Eighth cervical nerve root irritation.

a = Neck rigidity. Limited extension and rotation to the involved side.
b = Pain and paresthesia aggravated by coughing and sneezing.
c = Tenderness over exit of C_8.
d = Paresthesia and hypesthesia of inner forearm and little finger.
e = Subjective deep pain and tenderness from scapula down *inner* side of upper arm, inner forearm, to little finger.
f = Weakness of hand muscles.
g = No reflex changes.

the foramina close upon neck extension on the side toward which the neck rotates (Fig. 8–10). The dura has flexibility during all movement to avoid compression or traction (Cailliet, 1990).

Changes in the cervical spine from subluxation, disk herniation, or osteophyte formation may narrow the foramen (see Fig. 8–10) or cause soft tissue changes that encroach upon the cervical nerve root. Now the nerve roots in their dura no longer can escape compression or traction, and referred radicular pain results.

If the following can be elicited clinically, a cervical radiculopathic process manifested by upper extremity pain is suggested. The arm pain occurs from assumption of certain positions that increase the cervical lordosis. Usually these are neck extension with increased lordosis, thus closure of the foramina. Reading with bifocals, sitting at a word processor, and so forth are revealed in the history. Tension also causes increased closure of the cervical foramina, and this, too, can be confirmed by the history.

The examination reproduces the symptoms. Extending the neck with or without simultaneously rotating the neck also causes pain on that side. Lateral flexion compresses the ipsilateral nerve roots due to foraminal closure on that

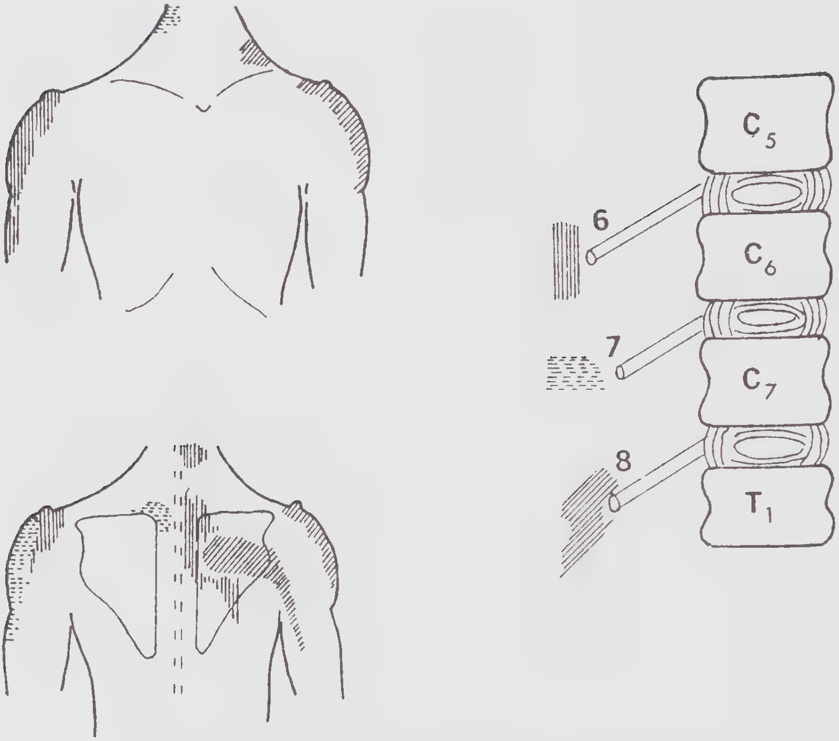


Figure 8–8. Regions of shoulder pain referral from cervical radiculitis. The areas depicted by hatching are the vague *shoulder area* referrals frequently complained of by patients. These are not the cervical root dermatomes, which are more specific in C_6, C_7, and C_8 and are referred to the hand, forearm, and fingers. The areas shown in this diagram may be derived from posterior primary rami, or sclerotomes, or they may be areas of myalgic tenderness.

side. The Spurling compression test results are often positive (Fig. 8–11), as are the manual traction and arm abduction tests for cervical radiculitis (Fig. 8–12). It is the sheath that is sensitive; it is a nociceptor site. Pressure or traction on the nerve root sheath (dura) can cause both local and referred pain. If it is suspected that the complaints of arm pain are cervically related, the pain can be reproduced by movement of the neck that encroaches on the nerve root or that places traction on the cervical nerve roots.

In cervical discogenic disease the intradiscal foraminal compression usually occurs between the fifth and sixth or sixth and seventh cervical vertebrae, with compression of the C_5 or C_6 nerve root. Pain or paresthesia is perceived down the lateral aspect of the arm and often into the fingers. If the lesion is a disk herniation with compression on the nerve root, flexion of the neck reproduces the

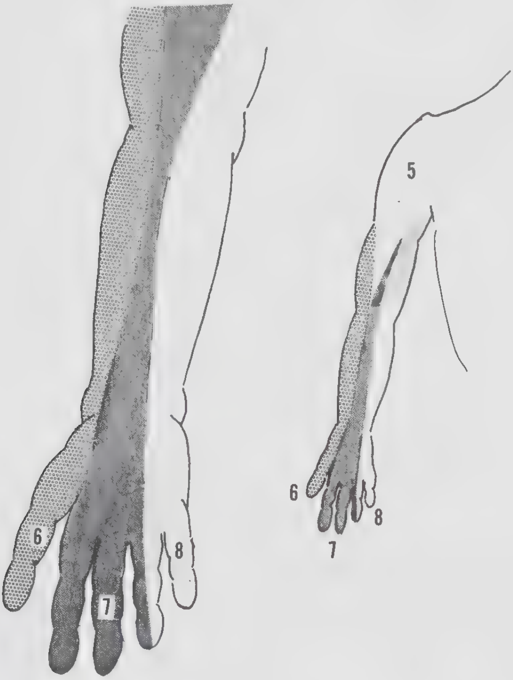


Figure 8–9. Dermatomes of cervical nerve roots C_5 to C_8.

referred symptoms. If the lesion is foraminal stenosis from degenerative spondylosis, neck extension reproduces the referred pain. Usually there are also symptoms referred to the neck, such as stiffness and limited movement.

The referral site in cervical radicular symptoms is often a dull ache in the interscapular region at the T_4, T_5, T_6 areas (Cloward).

Motion of the shoulder such as abduction, limited overhead elevation, limited weak external rotation by resistance to the rotator cuff muscles, and positive *drop test* results are not necessarily positive, and if so, they are superceded by eliciting cervically related symptoms. Because the scapular muscles are more active than the glenohumeral muscles in rotator cuff lesion, and because the scapular muscle originates in the lower cervical spine, it is apparent that there is a relationship between cervical radiculitis and glenohumeral lesion.

Diagnostic tests such as x-ray studies, MRI, CT scanning, or even myelography of the cervical spine may indicate pathologic process at various levels of the spine. Abnormal EMG study results may implicate nerve root involvement. It must be clearly stated, however, that the differential diagnosis between cervical referred pain and intrinsic shoulder pain is a clinical decision. Diagnosis of pain of a cervical nature may be confirmed by x-ray studies, but it is never the x-ray studies, per se, that are diagnostic.

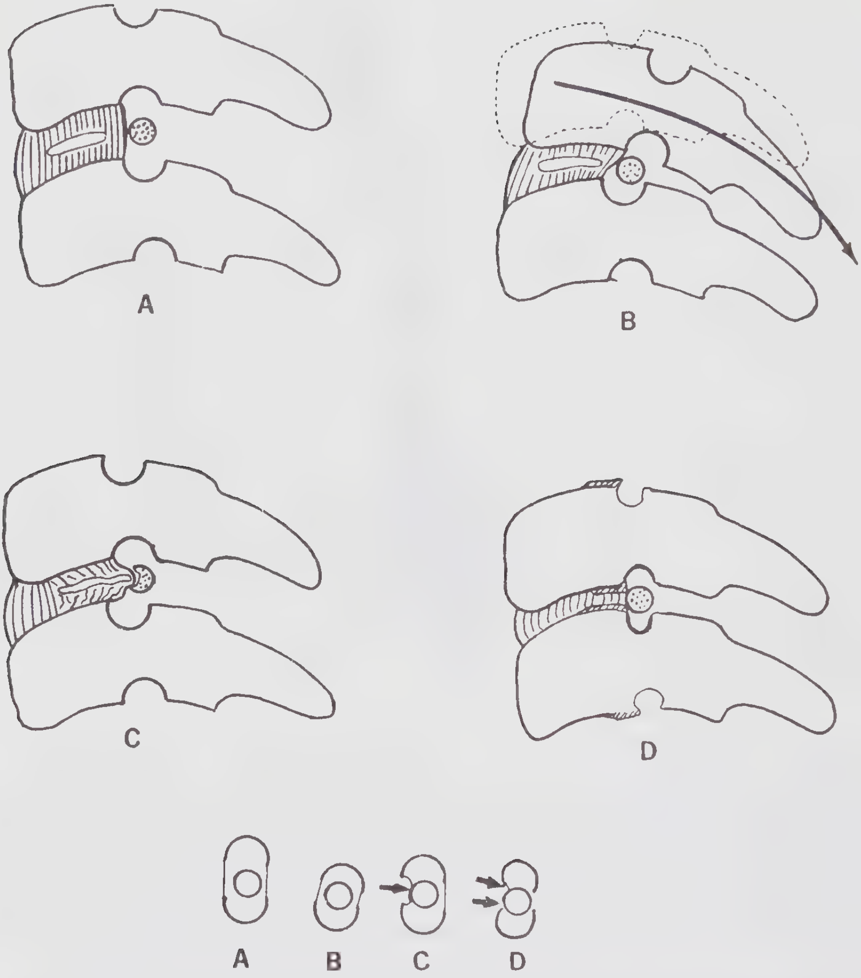


Figure 8–10. Foraminal opening variations. (*A*) Normal open intervertebral foramen with neck in neutral position, no rotation, and no lateral flexion. (*B*) Extension by backward gliding of the upper vertebra upon the lower vertebra normally narrows the foramen but does not compress the nerve root. (*C*) Compression of the nerve root by herniation of the intervertebral disk. (*D*) Distortion of the foramen by osteophytic changes of the joints of Luschka and disk degeneration.

THORACIC OUTLET SYNDROME

Another form of neurologically referred shoulder-arm pain can be from encroachment of neurologic tissues in the region of the shoulder girdle complex. The neurovascular elements responsible for symptoms in the upper extremity

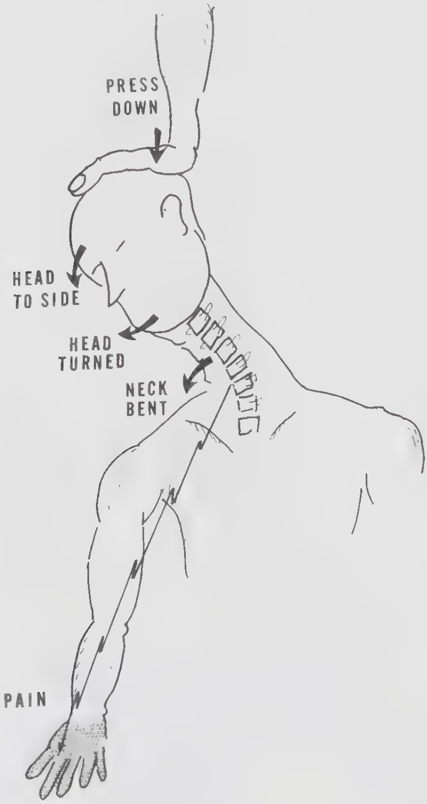


Figure 8–11. Spurling compression test for radiculitis. With the patient seated and the head turned and laterally flexed to the side of the radiculitis, there is downward compression upon the head. A *positive* test result reproduces the radicular symptoms.

exit at the root of the neck in their progression to the upper extremity through a limited space termed the *thoracic outlet.* Any constriction of this outlet may lead to painful disabling symptoms.

Within this outlet are found the brachial plexus, the subclavian artery, and the subclavian vein. Compression of any of these structures can produce symptoms of pain, paresthesias, swelling, temperature changes, and motor weakness, which affect the shoulder, arm, forearm, and hand.

The *neurovascular bundle,* which is the collective term for all the exiting nerves and vessels through this outlet (Fig. 8–13), passes through a series of narrow rigid spaces wherein the slightest anatomic or physiologic deviation can result in compression with resultant symptoms. Some of the tissues that may encroach on the neurovascular bundle have been designated as cervical rib, fascial remnants, abnormality of the first thoracic rib, interscalene muscle *spasm* or fibrous contraction, thickening of the normal fascia from persistent mechanical irritation, and prolonged abnormalities of posture. *Mechanical* compression of the neurovascular bundle between the bony components of the outlet is also considered a predominant cause.

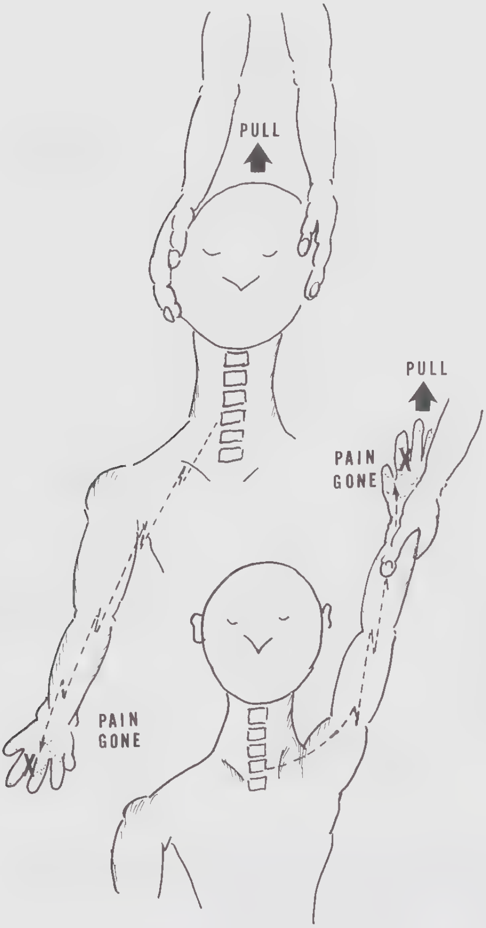


Figure 8–12. Manual traction and arm abduction test for cervical radiculitis. Upper figure depicts manual action upon the head. If there is disappearance or diminution of radicular symptoms, the test result is positive. Lower figure depicts elevation above the head of the arm of the side of the radiculitis. Disappearance of symptoms is a positive test result.

The resultant symptoms from encroachment on the bundle may be neurologic or vascular, dependent on which component of the bundle is under compression or traction.

The term *thoracic outlet* (also termed *cervical dorsal outlet*) *syndrome* was used synonymously with *scalene anticus syndrome* when neurovascular compression was attributed to spasm and shortening of the anterior scalene muscle (Naffziger and Grant). The thoracic outlet syndrome (TOS) was thought to be caused by tightening, sustained contraction, fibrosis, or hypertrophy of the anterior scalene muscle.

Inasmuch as the fascia has been incriminated as a factor in symptomatic TOS, it merits description. The prevertebral fascia is a firm membrane lying anteriorly to the prevertebral muscle (longus cervicis; longus capitis; anterior, middle, and posterior scalenes; and the rectus capitis muscles) (Fig. 8–14). The fas-

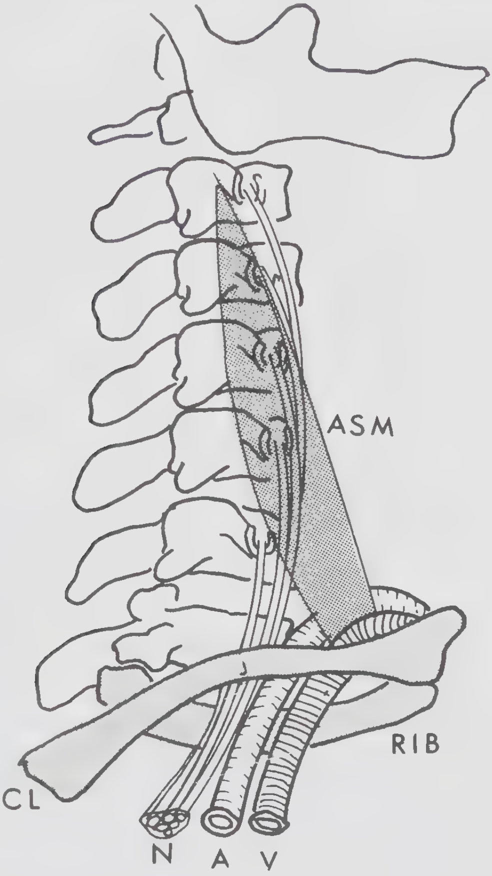


Figure 8–13. Thoracic outlet. Schematic depiction of the course of the brachial plexus subclavian bundle from the cervical spine over the first rib, and under the clavicle (CL) with the relationship to the anterior scalene muscle (ASM). (A = subclavian artery; N = brachial plexus; V = subclavian vein.)

cia is attached to the base of the skull just anterior to the capitis muscles and travels downward and laterally ultimately to blend with the fascia of the trapezius muscle. In its course it envelops the scalene muscles and also binds down the subclavian artery and the three trunks of the brachial plexus.

The prevertebral fascia is firmly adherent to the anterior aspect of the cervical vertebrae and and clavicle. It proceeds medially to attach to the transverse processes (Fig. 8–15) of the cervical vertebrae and in its course covers all the cervical nerve roots. The fascia *does not* ensheathe the subclavian or axillary veins and therefore cannot cause venous congestion, but with all the other nerves and vessels so ensheathed, it is apparent that constriction of these vital structures is possible.

Because the nerves of the sympathetic outflow from the stellate ganglia penetrate this fascia, it stands to reason that fibrous constriction can entrap or at least irritate the sympathetic nerves and cause symptoms attributable to sympathetic nerve stimulation.

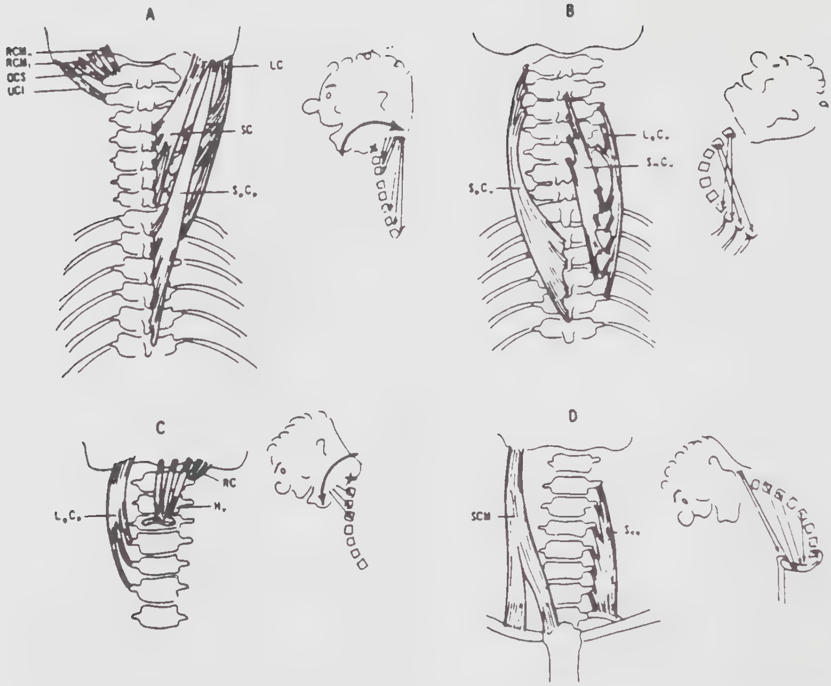


Figure 8–14. Musculature of the head and neck. *(A)* and *(B)* The musculature of the extensor mechanism of the head and neck. *(A)* The capital extensors attach to the skull and move the head upon the neck. *(B)* The cervical extensors originate and attach upon the cervical spine and alter the curvature of the cervical spine. *(C)* and *(D)* Flexion musculature. *(C)* The capital flexors flex the head upon the neck. *(D)* The cervical flexors attach exclusively upon cervical vertebrae and have no significant functional attachment to the skull.

RCM_n = rectus capitis minor
RCM_j = rectus capitis major
OCS = obliquus capitis superior
OCI = obliquus capitis inferior
$\mathrm{L}_g\mathrm{C}_p$ = longus capitis
RC = rectus capitis anterior and lateral
H_y = hyoideus and suprahyoid muscles

LC = longissimus capitis
SC = semispinalis capitis
$\mathrm{S}_p\mathrm{C}_p$ = splenius capitis
$\mathrm{S}_p\mathrm{C}_v$ = splenius cervicis
$\mathrm{L}_m\mathrm{C}_v$ = longissimus cervicis
$\mathrm{S}_m\mathrm{C}_v$ = semispinalis cervicis
SCM = sternocleidomastoid
S_{ca} = scalene medius and anticus

Symptoms of TOS

Numbness, tingling, pain, and paresthesias are the most common complaints. The characteristics of this complaint are variable, and often the patient cannot *specifically* locate the complaints.

The symptoms are often generalized and may include all of the upper ex-

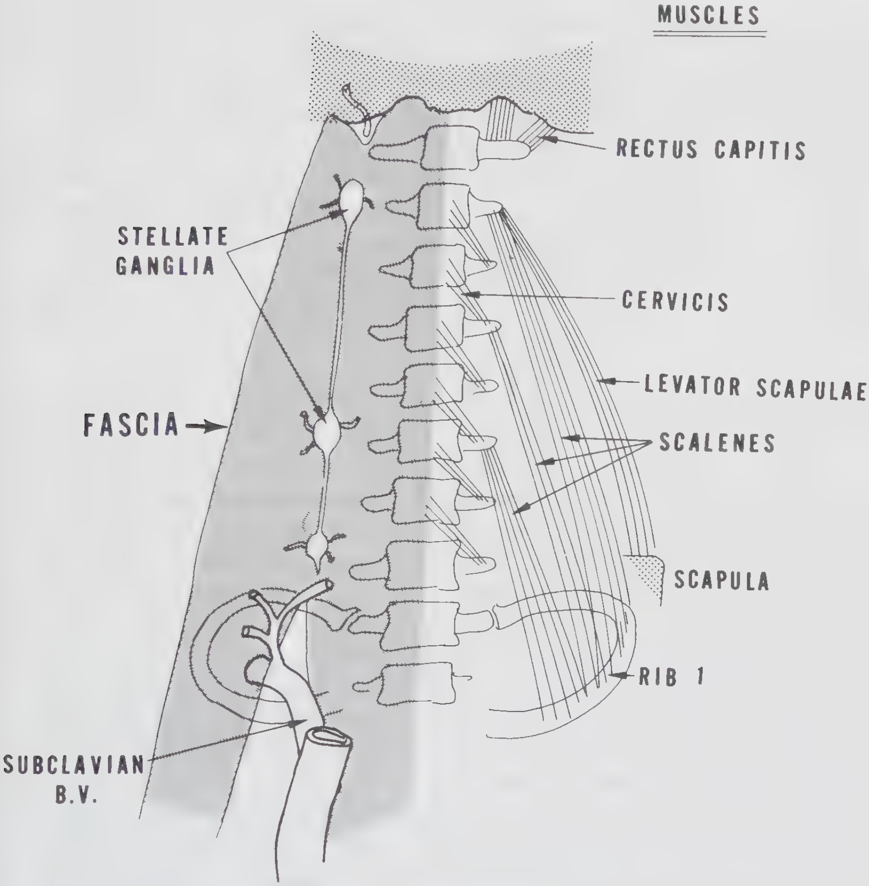


Figure 8–15. Anterior view of *prevertebral fascia* and muscles. The fascia is a firm membrane in front of the prevertebral neck muscles. It binds these muscles to the subclavian artery and three trunks of the brachial plexus. All the cervical nerve roots are beneath the fascia. The fascia is attached to the anterior margin of the cervical vertebral bodies and to the clavicle. The cervical sympathetic trunk lies in front of the fascia.

tremity as well as the shoulder and scapular area. From a careful detailed history the symptoms are usually related to a particular position or movement. A pattern of severe pain upon arising in the morning may incriminate the sleeping position wherein the person sleeps with the arms overhead or folded beneath the head and thus serving as a pillow. Occurrence during the day may also be related to the arm positions assumed during the daily occupation. Reproduction of the symptoms is a major part of the examination, indicating the position ascertained as the causative factor in activities of daily living.

The medial cord, the most inferior portion of the brachial plexus, is most vulnerable to compression or angulation. Symptoms, therefore, are distributed mostly from C_8 to T_1. These nerves subserve the medial brachial and medial antebrachial cutaneous nerves and the ulnar nerve. They implicate C_8 dermatomal sensation of the medial aspect of the arm and forearm and the fifth and ring fingers as well as the hypothenar eminence.

Compromise of the arterial (subclavian) aspect of the bundle produces symptoms of coldness, weakness, cyanosis, and pallor. Rarely, gangrenelike symptoms and ultimately an objective finding may be produced, but usually the subjective findings are complained of by the patient and reproduced by the examiner.

Cervical Ribs

Supernumerary ribs (cervical ribs) arising from the lower cervical vertebrae, usually the seventh cervical vertebra, have been considered a cause of neurovascular compression at the thoracic outlet (Tyson and Kaplan). These ribs can be anything from a brief stub—an elongation of the transverse process—to a complete rib uniting with the first rib with a cartilage superimposed. Because not all cervical ribs are calcified, not all appear on routine x-ray studies.

These ribs are associated with numerous fibrous bands of the prevertebral fascia, and it is these fibrous bands that have been considered to cause TOS. Symptoms alone of TOS are not diagnostic of cervical ribs. The symptoms are reproduced by TOS examination, and the ribs are discovered by x-ray studies or during surgical intervention.

An anomaly of the first thoracic rib has also been implicated in TOS. This rib originates from the first thoracic vertebra and may encroach upon the lower component of the brachial plexus. It, too, cannot be clinically diagnosed by TOS maneuvers but is discovered during x-ray studies or surgical intervention when the history and physical examination suggest TOS.

Scalene Anticus Syndrome

The scalene muscles were originally considered the cause of TOS and were so claimed by Adson and Coffey and highlighted by Naffziger and Grant. The subclavian artery usually courses between the anterior and middle scalene muscles accompanied by the brachial plexus. This vessel may pass anterior to the anterior scalene muscle or pass through its belly, but this is rare and is discovered also only during surgical intervention.

The scalene muscles originate from the transverse processes of the cervical vertebrae and insert over a variable width of the first thoracic rib. This V-shaped space between the first rib and the scalenes is termed the *thoracic*, or *cervical, outlet*.

Claviculocostal Syndrome

Because the clavicle and the first rib are parallel to each other and form a portion of the thoracic outlet, compression between these two structures is implicated in clinical TOS and termed *costoclavicular compression syndrome*. This compression is confirmed clinically during examination after eliciting symptoms of TOS by the specific maneuver.

Hyperabduction TOS Syndrome

Lastly, *hyperabduction compression syndrome* is a form of TOS in which the symptoms allegedly are reproduced by actively compressing the neurovascular bundle between the first rib and the clavicle by assuming a position of extreme posterior flexion and hyperabduction of the shoulder girdle.

Other rare causative factors can compress the neurovascular bundle, such as congenital or acquired factors of the rib or clavicle or abnormalities of the subclavian vessels or scalene muscles. These are discovered in evaluation and exploration of the outlet when the symptoms suggest neurovascular compression.

Physical Examination

Objective documentation of the specific dermatome(s) involved can be documented by light touch, pinprick, or scratch. Wasting and atrophy of the hypothenar and ulnar intrinsic musculature can be noted in long-lasting cases. Fasciculation can be noted with nerve compression.

Usually the diagnosis of TOS is made by reproducing the symptoms clinically. The following syndromes are diagnosed and confirmed by reproducing the symptoms using the tests or maneuvers described below.

Scalene Anticus Syndrome and the Adson Maneuver. The patient fully extends (posteriorly flexes) the head and neck, turns the chin toward the side of the symptoms, and holds the breath after a deep inspiration. If the pulse becomes obliterated when it is simultaneously palpated, it is a *positive* test result. Because the pulse can be obliterated in many normal asymptomatic persons, it is the reproduction of the symptoms that determines the diagnosis.

The mechanism that explains the Adson test is that the extension and turning of the head causes elongation of the scalenes and their prevertebral fascia and thus compresses the neurovascular bundle. The inspiration invokes the respiratory effect of the scalenes on the rib cage inasmuch as they are accessory respiratory muscles (Fig. 8–16).

It must be remembered that cervical radiculopathy, compression of nerve

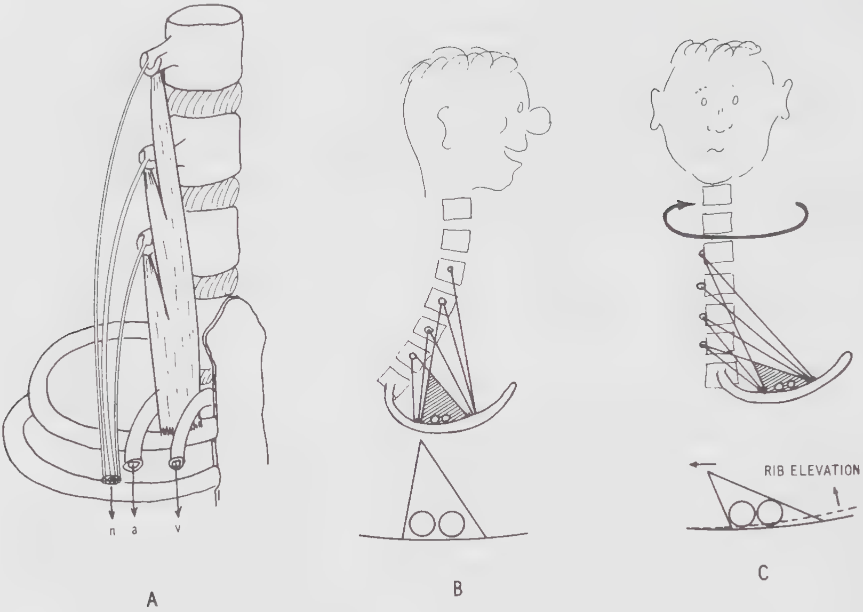


Figure 8–16. Scalene anticus syndrome. *(A)* Relationship of the neurovascular bundle. The subclavian artery (a) passes behind the anterior scalene muscle, loops over the first rib, and is joined by the brachial plexus (n). The artery is separated from the subclavian vein (v) by the anterior scalene muscle. The median scalene muscle (not shown) lies behind the nerve (n). *(B)* The triangle formed by the scalenes and the first rib. *(C)* Distortion from turning the head toward the symptomatic side. Compression of the neurovascular bundle—the brachial plexus, the subclavian artery, and occasionally the subclavian vein—can be pictured from the test maneuver of the anticus scalene syndrome.

roots as they emerge from their foramina, can also be reproduced by extending and rotating the neck to the ipsilateral side (Cailliet), causing dermatomal subjective and objective encroachment. The Adson maneuver must be evaluated critically before implicating dermatomal and myotomal from encroachment on the thoracic outlet and not on the cervical foramenal sites.

Claviculocostal Syndrome. Presence of the *claviculocostal syndrome* is ascertained by performing the *costoclavicular maneuver* (Fig. 8–17). This requires placing the patient in an exaggerated military position with the shoulder posteriorly *braced* and depressed. This exaggerated position depresses the clavicle on the first rib and compresses the neurovascular bundle. The pulse(s) are obliterated, and the symptoms of paresthesia are reproduced.

Pectoralis Minor Syndrome and Test. The test for *pectoralis minor syndrome* (*B* in Fig. 8–17), a maneuver that can reproduce TOS, is merely an

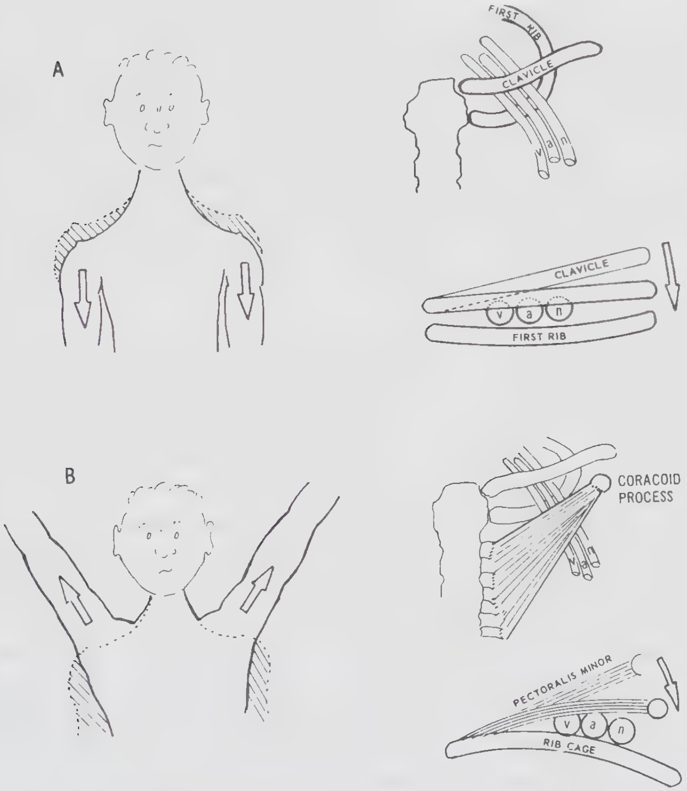


Figure 8–17. Claviculocostal and pectoralis minor syndromes. *(A)* Claviculocostal syndrome. The neurovascular bundle is compressed between the clavicle and the first rib by retraction and depression of the shoulder girdles. *(B)* Pectoralis minor syndrome. The neurovascular bundle may be compressed between the pectoralis minor and the rib cage by elevating the arms in a position of abduction and moving the arms behind the head.

abduction of the arms and a retracting of them posteriorly downward. This maneuver is essentially a modification of the hyperabduction test.

Treatment

Conservative nonsurgical treatment must be thoroughly pursued before any surgical intervention is contemplated, unless there are significant progressive *objective* neurologic or vascular signs.

Educating the patient on the mechanical basis of the symptoms in a manner the patient can understand will ensure greater acceptance of and adherence to a program. This is better than merely giving the patient a list of exercises that may or may not be performed effectively.

Correcting posture, as has been stated in Chapter 5, is the major component of therapy. This indicates proper posture in sitting, standing, walking, and in every daily activity. It implies that the patient must fully understand that good posture is constant, not something assumed merely during the concentrated exercise; it must become a matter of daily unconscious habit.

When symptoms can be reproduced, the offending position and/or posture can be brought immediately to the patient's attention to educate him or her on the rationale of corrective therapy.

Flexibility exercises are valuable, but which tissue(s) must be made *flexible* demands careful evaluation and precise physical therapy of stretch with or without spray (Travell and Simons). A daily home exercise program enhances the assurance of increased flexibility. Strengthening and improving endurance of the scapular elevators are considered valuable in correcting posture and relieving symptoms of TOS (Fig. 8–18).

Mechanical or manual traction has limited value, as does the use of a collar, for ensuring correct posture. A cervical pillow may afford relief of the patient who awakens with TOS symptoms.

Emotional correction must be entertained when tension, anxiety, depression, and/or anger is considered a major or contributing factor. *Stress management* has become a valuable adjunct to persistent postural tension TOS.

Operative intervention is indicated when there is confirmed evidence of objective TOS that is failing to respond to appropriate conservative management for a significant period of time or when there may be objective neurologic and/or vascular findings. Objective evidence of neurologic impairment can be documented by EMG studies (Urschel and associates) and conduction velocity and cortically evoked potential studies, which enhance the advisability of surgical intervention over clinical diagnosis with the attendant possible surgical failure (Derkash and colleagues).

BRACHIAL PLEXUS INJURIES

In any shoulder trauma that causes subluxation or luxation of the glenohumeral joint or a fracture-dislocation, the brachial plexus (Fig. 8–19) can be traumatized. Even in a clavicular fracture the brachial plexus might sustain injury. Severe sports injury to the shoulder can cause a traction injury to the brachial plexus. It has been reported (Kaplan and Tanner) that some stroke patients with a flaccid extremity sustained traction injuries to the brachial plexus.

The history of trauma followed by commensurate symptoms and neurologic symptoms indicates plexus injury. Acute injury followed by severe upper extremity pain may occur with an initial paucity of objective neurologic findings. There is tenderness upon deep pressure over the neurovascular fossa and the

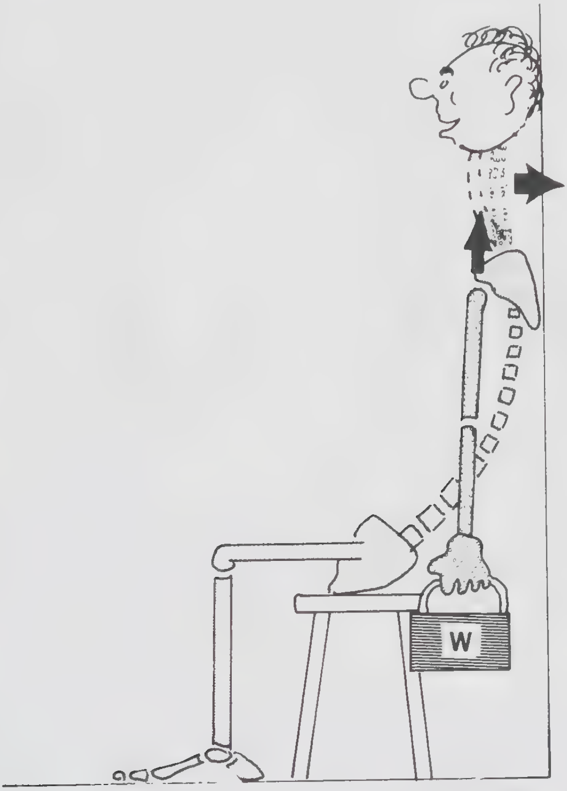


Figure 8–18. Posture-scapular elevation exercises. Patient is seated with back to wall, head and neck pressed against the wall, which decreases the cervical lordosis. With arms fully extended and dependent, weights are lifted in a shrugging motion. Weights vary from 5 to 30 pounds.

scalene muscles. Pain can be reproduced or aggravated by distraction of the neck away from the shoulder.

Objective findings will reveal sensory and motor involvement of many roots in contradiction to a cervical root involvement, which implies only a single nerve root. The roots involved usually encompass the roots from C_5 to T_1, with all the dermatomes and myotomes involved. In medical literature there have emerged two classic neurologic patterns: Erb's palsy (the flail arm), which is an upper trunk lesion (that is, C_5 to C_6) and the Klumpke's paralysis from involvement in the lower trunk of the plexus (C_8 to T_1).

Neurologic examination will reveal hyalgesia, hypesthesia, and multiple motor paresis. Ultimately EMG findings will confirm the diagnosis—the determination of which aspect of the brachial plexus has sustained injury. But it must be remembered that a period of 3 weeks may pass before significant demyelination is appreciated on the EMG.

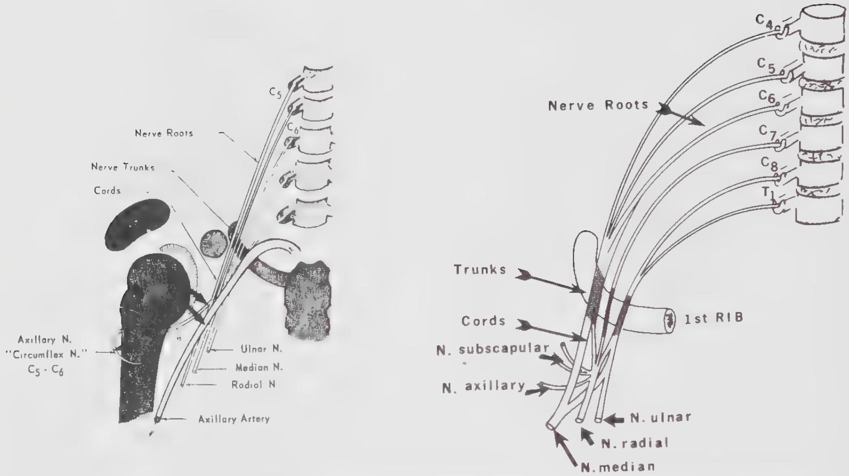

Nerve Roots
Nerve Trunks
Cords
C_5
C_6
Axillary N.
"Circumflex N."
C_5 - C_6
Ulnar N.
Median N.
Radial N
Axillary Artery
C_4
C_5
C_6
C_7
C_8
T_1
Nerve Roots
Trunks
Cords
1st RIB
N. subscapular
N. axillary
N. ulnar
N. radial
N. median

Figure 8–19. Schematic brachial plexus. Emphasis *(shaded area)* is placed on the posterior cord forming the radial, axillary, and subscapular nerves, this being the cord most often involved in shoulder separation.

Besides traction injury to the brachial plexus, in a severe trauma there may be injury to the axillary blood vessels. When this occurs, the hand becomes pale, blue, then cold. There is a pulse deficit and possibly axillary swelling or ecchymosis. This constitutes an emergency requiring urgent surgical repair.

Brachial Plexus Neuritis

A condition that has been called *brachial plexus neuropathy* has also been termed *localized neuritis of the shoulder, acute brachial radiculitis, localized nontraumatic neuropathy, acute shoulder neuritis, serum neuritis* (Martin and Kraft), *paralytic brachial neuritis*, and *neuralgic amyotrophy*. These terms have evolved in contradiction to mechanical TOS, in that they have been determined to be inflammatory and/or infectious in their origin.

The initial presenting symptom is shoulder pain with ultimate weakness of specific muscles of the involved plexus innervation. Clinically there is a rapid onset of severe pain, usually followed by weakness and paresis. The latter often occurs within 2 weeks of the onset of pain. The pain is usually intense and is described as sharp, stabbing, throbbing, or deep aching. Usually there is also an associated muscle soreness (Tsairis and associates).

Pain occurs predominantly in the upper plexus and is rarely noted below the elbow. In half of the diagnosed patients the symptoms are unilateral.

Currently this is considered to be a neuropathy associated with a systemic infectious disorder, or it is an allergic hypersensitivity reaction to a systemic disease process. This syndrome occasionally occurs after administration of a foreign serum, such as a vaccine. In many cases the etiologic factor remains undiscovered.

Neurologic findings reveal that complete bilateral plexus involvement is rare. Unilateral total plexus involvement with spotty contralateral involvement has been reported. The axillary and the suprascapular nerves—thus the deltoid and the spinati muscles—are the most frequently afflicted, with one shoulder or both shoulders involved. Winging of the scapula is frequent.

Fasciculation is a common observation, and atrophy frequently evolves in the afflicted muscles. Sensory loss is observed in 80 percent of cases, as is diminution of deep tendon reflexes.

Confirmatory studies are sparse. Immunologic studies reveal no specific abnormalities, and x-ray studies, including myelography, are noninformative. Studies of cerebrospinal fluid (CSF) are essentially normal. Electromyographic nerve conduction studies are diagnostic of progressive denervation and assist in the identification of the specifically involved nerve(s).

Treatment is nonspecific; it is merely supportive and essentially meant to offer relief from pain during the acute phase. Rehabilitation for regaining specific muscle group strength may be indicated during the recovery phase. Happily, the prognosis is excellent, and the vast majority of patients make a complete or functionally beneficial recovery.

SUPRASCAPULAR NERVE ENTRAPMENT

The suprascapular nerve originates in C_5 to C_6 and unites to form a single nerve rather than to become a branch of the brachial plexus (Fig. 8–20). It passes behind the plexus to the upper border of the scapula and proceeds through the suprascapular notch. While within this notch it is passed over by the transverse scapular ligament. It then enters the supraspinatus fossa to innervate the supraspinatus and infraspinatus muscles and to carry sensation to the shoulder joint capsule and the acromioclavicular joint.

The suprascapular nerve is essentially a motor nerve, and its impairment causes paresis. Because it is also a sensory nerve to the shoulder joint, it is the site of a chemical nerve block in the event of an intractable painful shoulder.

The nerve normally becomes fully stretched when the arm is held across the chest in extreme adduction. Any further adduction past the physiologic limits causes traction upon the nerve. Impairment of this nerve thus can occur from an athletic injury, industrial insult, or a fall that causes the arm to be adducted abruptly and excessively.

The history depicts the injury. Pain may be noted, but usually the injury is followed by evidence of paresis of the supraspinatus muscle. The overall picture has been thoroughly described in the chapter on shoulder injury, depicting the shrugging mechanism and the drop arm test, but suffice it here to repeat: The

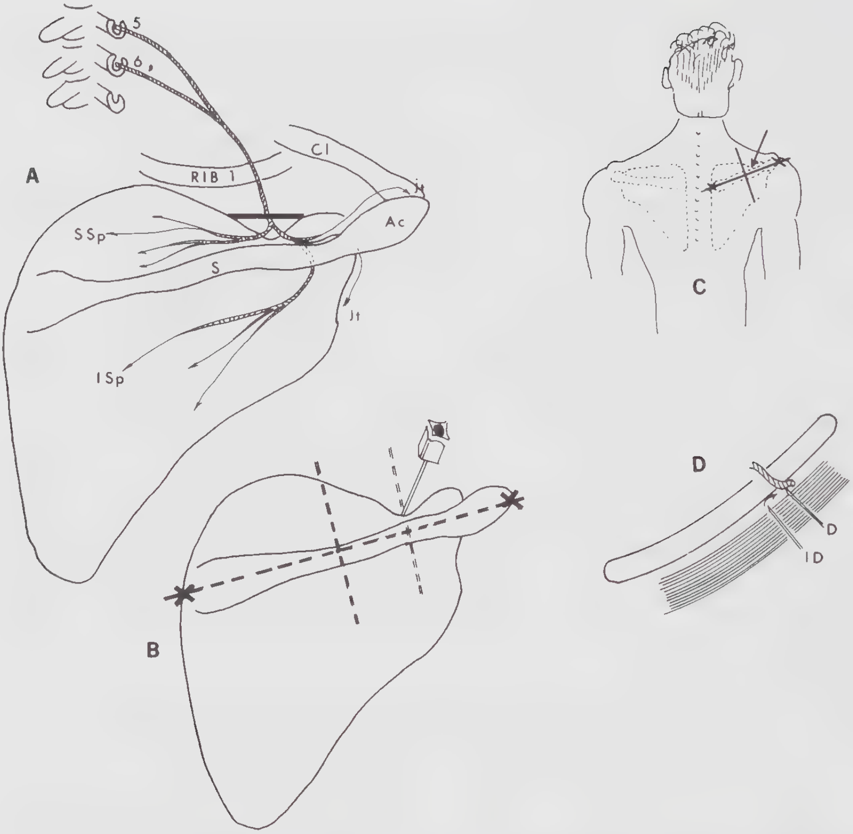


Figure 8–20. Suprascapular nerve block. *(A)* The anatomy and course of the suprascapular nerve, originating from C_5 to C_6. The motor nerve to the supraspinatus (SSp), and infraspinatus (ISp) and the sensory branches to the acromioclavicular joint and the shoulder joint (jt) are shown. *(B,C)* Bisection of a line drawn along the scapular spine with the site of the groove and of needle insertion. *(D)* The difference between direct nerve contact and indirect (ID) by infusion along the posterior portion of the scapula below the supraspinatus muscle.

arm cannot be abducted because the beginning of the scapulohumeral abduction is impaired. The deltoid muscle normally cannot function until the arm has been abducted by the external rotator (the supraspinatus). Thus it appears, clinically, that there is rotator cuff failure in a suprascapular nerve injury, as well as in a complete rotator cuff tear. This condition must always be differentially considered in an injured arm that presents as a total rotator cuff tear.

Definitive diagnosis may await EMG verification of denervation of the supraspinatus and infraspinatus muscles. There is a delay of 21 days, however, before frank denervation is seen on EMG, which may delay specific diagnosis.

Treatment is supportive until regeneration ensues. Physical therapy exercises to strengthen the muscles to ensure adequate recovery is indicated. Shoulder separation (subluxation) frequently occurs, and if this is symptomatic, it can be addressed by an appropriate sling.

DORSAL SCAPULAR NERVE ENTRAPMENT

The dorsal scapular nerve innervates the rhomboid muscles. This nerve originates from the C_5 root and, shortly after its origin, perforates the median scalene muscle. If it becomes irritated or entrapped in its passage, it affects the motor function of the rhomboid muscles. An injury to this nerve can be a fall or a blow upon the scapula that causes stretching of the scalene muscles or a severe depression of the scapula. A neck injury can initiate this syndrome.

Pain may be felt as a dull, deep, and vague ache in the region of the medial edge of the scapula. Because this nerve is principally a motor nerve, painful sensation may be absent, and only paresis of the rhomboid may be noted. The function of the rhomboid muscles is scapular downward rotation and depression.

The clinical suspicion can be verified by specific EMG study. Treatment is supportive, but when entrapment symptoms of either paresis or severe pain are persistent, surgical neurolysis is indicated (Kopell and Thompson).

BIBLIOGRAPHY

Adson, AW: Cervical ribs: Symptoms, differential diagnosis for section of the insertion of the scalene anticus muscle. Journal of the International College of Surgeons 16:546, 1951.

Adson, AW and Coffee, JR: Cervical rib: Method of anterior approach for relief of symptoms by division of scalene anticus. Ann Surg 85:839, 1927.

Aymond, JK, Goldner, JL, and Hardaker, WT: Neuralgic amyotrophy: A review paper: Orthopedic Review vol 17 (12):1275, 1989.

Cailliet, R: Neck and Arm Pain, ed 3. FA Davis, Philadelphia, 1990.

Cailliet, R: Soft Tissue Pain and Disability, ed 2. FA Davis, Philadephia, 1988.

Cloward, RB: Cervical diskography: a contribution to the etiology and mechanism of neck, shoulder, and arm pain. Ann Surg 150:1052, 1959.

Derkash, RS, et al: The results of first rib resection in thoracic outlet syndrome. Orthopedics 4 (9):1025, 1981.

Gage, M and Parnell, H: Scalene anticus syndrome. Am J Surg 3:252, 1947.

Haymaker, W and Woodhall, B: Peripheral Nerve Injuries: Principles of Diagnosis, ed 2. WB Saunders, Philadelphia, 1953.

Kaplan, PE and Tanner, ED: Musculoskeletal Pain and Disability. Appleton & Lange, Norwalk, CT, 1989, p 104.

Kopell, HP and Thompson, WA: Peripheral Entrapment Neuropathies, ed 2. RE Krieger, Huntington, NY, 1976.

Kraft, GH, Johnson, EW, and LaBan, MM: The fibrositis syndrome. Arch Phys Med Rehabil 49:155, 1968.

Lloyd, JW and Rosati, LM: Neurovascular compression syndrome of the upper extremity. Clin Symposia 10, 1958.

Martin, WA and Kraft, GH: Shoulder Girdle Neuritis: A Clinical and Electrophysiological Evaluation. Milit Med 139(1):21, 1971.

Naffziger, HC and Grant, WT: Neuritis of the brachial plexus mechanical in origin: The scalene syndrome. Surg Gynecol Obstet 67:722, 1938.

Ross, DB: Congenital anomalies associated with thoracic outlet syndrome. Am J Surg 132:771, 1976.

Tsairis, PT, Dyck, PJ and Mulder, DW: Natural History of Brachial Plexus Neuropathy. Arch Neurol 27:109, 1972.

Travell, JG and Simons, DG: Myofascial Pain and Dysfunction: The Trigger-Point Manual. Williams & Wilkins, Baltimore, 1983.

Tyson, RR and Kaplan, GF: Modern Concepts of Diagnosis and Treatment of the Thoracic Outlet Syndrome. Symposium on Surgery of the Shoulder. Orthop Clin North Am 6(2):507, 1975.

Urschel, HC et al: Objective diagnosis (ulnar nerve conduction velocity) and current therapy of the thoracic outlet syndrome. Ann Thorac Surg 12(6):608, 1971.

Urschel, HC and Razzuk, MA: Current Concepts: Management of the Thoracic-Outlet Syndrome. N Engl J Med 286(21):1140, 1972.

Woods, WW: Thoracic Outlet Syndrome. West J Med 128:9, 1978.

Woods, WW: Personal Experiences with Surgical Treatment of 250 Cases of Cervicobrachial Neurovascular Compression Syndrome. Journal of the International College of Surgeons 44(3):273, 1965.

CHAPTER 9

The Shoulder in the Hemiplegic Patient

THE COMPLETED STROKE

The patient afflicted with the *completed stroke,* the hemiplegic patient, frequently has as a residual impairment a painful, nonfunctioning or only partially functioning shoulder, and he or she may also have an impending or occurring shoulder-hand-finger syndrome of reflex sympathetic dystrophy.

The entire stroke sequelae must be neurologically evaluated (Fig. 9–1), and the role of the shoulder in this neurologic disease entity must be understood. Appropriate treatment demands a neuro-orthopedic understanding of the functionally impaired shoulder.

The four stages usually experienced by the patient suffering from a cerebrovascular incident are the transischemic attack stage, the flaccid stage, the spastic stage, and the synergy stage. (Fig. 9–2).

Transient Ischemic Attack (TIA)

The ischemic attack (TIA) is a cerebrovascular incident with a degree of neurologic impairment *lasting less than 24 hours;* there is complete functional restoration of neuromuscular activities.

Because there are no residual neurologic disabilities, no treatment to rehabilitate the patient is required. Care of the patient with a confirmed TIA concerns the contributing factors, such as hypertension, diabetes, smoking, and family history. Prevention of recurrence or progression into a *completed stroke* is the goal.

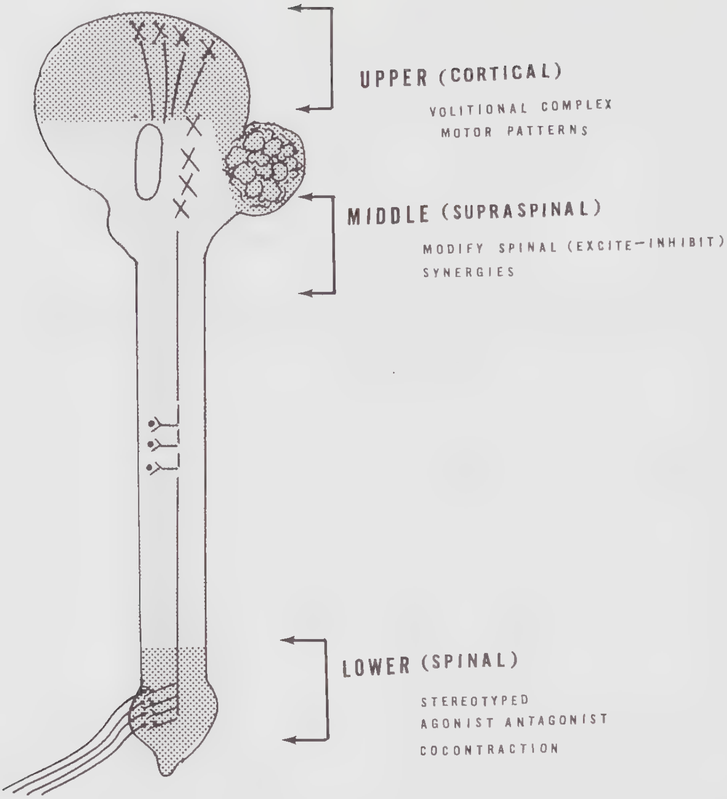

UPPER (CORTICAL)
VOLITIONAL COMPLEX MOTOR PATTERNS
MIDDLE (SUPRASPINAL)
MODIFY SPINAL (EXCITE–INHIBIT) SYNERGIES
LOWER (SPINAL)
STEREOTYPED AGONIST ANTAGONIST COCONTRACTION

Figure 9–1. Central nervous system. (From Cailliet, page 3, with permission.)

Some residual neurologic *signs* may remain, such as a Babinski or Hoffman sign or some increased muscular tone, but no significant functional loss remains.

When there has been a cerebrovascular episode with continuation of neurologic impairment persisting longer than 24 hours, the condition is termed a *completed stroke.* The persistent neurologic impairment proceeds, beginning with the flaccid stage.

Flaccid Stage

The flaccid stage is one of areflexia: total loss of deep tendon reflexes and loss of tone of the afflicted side. The involved extremity or extremities are flaccid. There is no specific motor function that can be initiated by the patient. Sensory loss is possible and variable, but when carefully evaluated it is found to be diminished.

Progression of this stage is either recovery—total or partial—or progres-

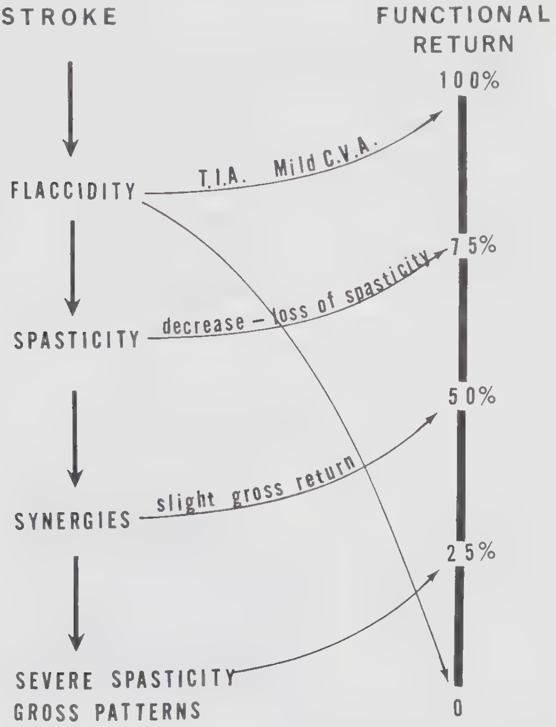

STROKE
FUNCTIONAL RETURN
100%
FLACCIDITY
T.I.A. Mild C.V.A.
75%
SPASTICITY
decrease – loss of spasticity
50%
SYNERGIES
slight gross return
25%
SEVERE SPASTICITY
GROSS PATTERNS
0

Figure 9–2. Estimate of spontaneous recovery from stroke. (From Cailliet, page 5, with permission.)

sion to the next, *spastic*, stage. Duration of this flaccid stage varies from seconds to months. The extent of this flaccidity is also variable, from a total extremity to a segment of an extremity. Duration longer than 2 weeks has been considered as prognostically unfavorable.

Involvement of the shoulder during this stage is important in regard to the desired therapeutic approach and in prognosticating the recovery of the upper extremity. It bears repeating that hand function demands proper shoulder motion to place the hand in a functional position for activities of daily living.

Spastic Stage

The flaccid extremity may have made complete recovery or it may have remained in a permanent flaccid condition, but usually in a completed stroke there is progression to the spastic stage.

The neurologic component of these stages is evidenced by the fact that the midbrain, pons, and medulla contain the flexor patterns of the upper extremity and the extensor patterns of the lower extremities, which now control the neuromuscular function of the body part involved (Fig. 9–3). Normally the neocortex exerts control of these basic patterns and refines their coordinated function (see Fig. 9–2). Each area of the premotor cortex controls a specific region of the musculature, including the extremities, the trunk, the face, pharynx, and glottis (Fig. 9–4). A larger area of the premotor cortex is devoted to speech, oral functions, and the hand, especially the thumb (Fig. 9–5).

In the patient with a stroke the upper connections to the midbrain are sev-

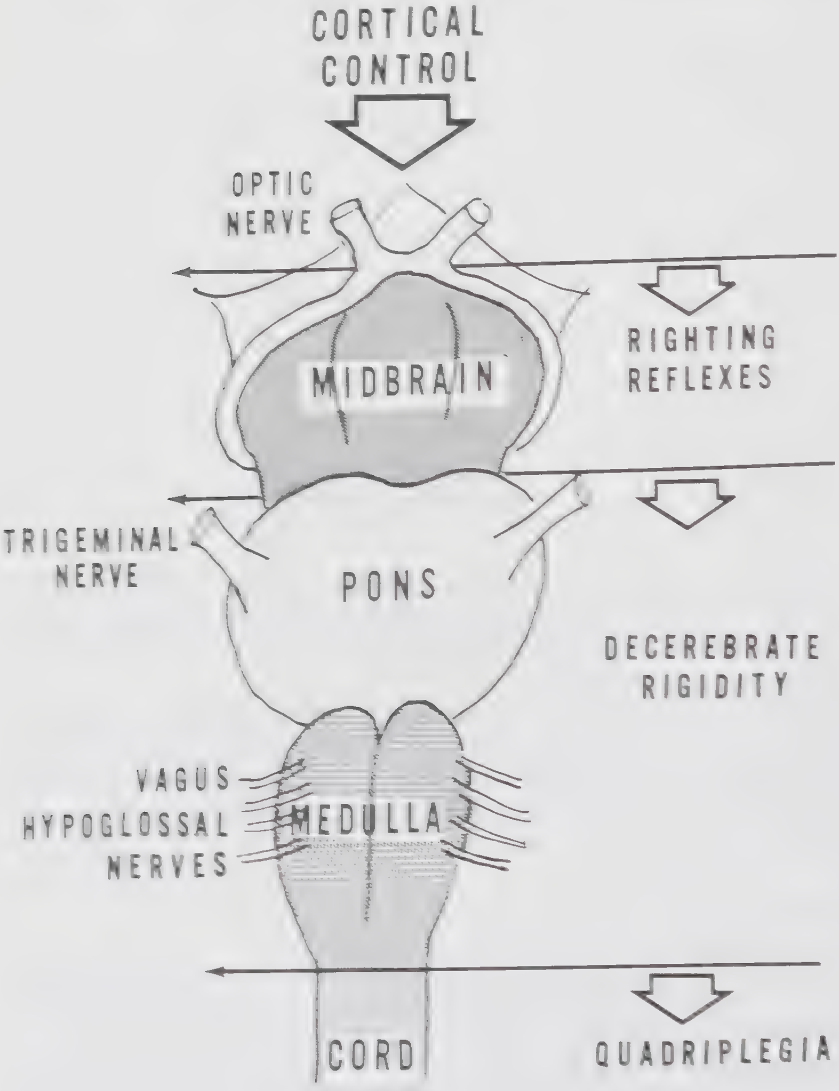

CORTICAL CONTROL
OPTIC NERVE
RIGHTING REFLEXES
MIDBRAIN
TRIGEMINAL NERVE
PONS
DECEREBRATE RIGIDITY
VAGUS
HYPOGLOSSAL
NERVES
MEDULLA
CORD
QUADRIPLEGIA

Figure 9–3. Midbrain, pons, and medulla.

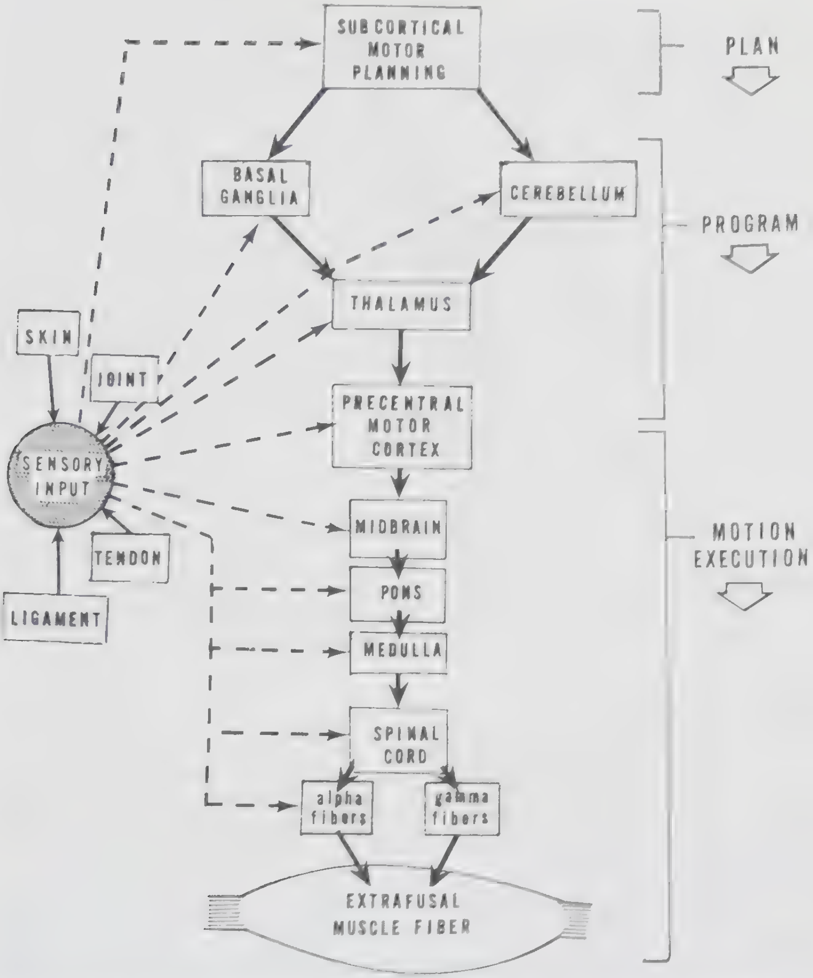

SUBCORTICAL MOTOR PLANNING
PLAN
BASAL GANGLIA
CEREBELLUM
PROGRAM
THALAMUS
SKIN
JOINT
PRECENTRAL MOTOR CORTEX
SENSORY INPUT
MIDBRAIN
MOTION EXECUTION
TENDON
PONS
LIGAMENT
MEDULLA
SPINAL CORD
alpha fibers
gamma fibers
EXTRAFUSAL MUSCLE FIBER

Figure 9–4. Spinal and supraspinal motor centers. (Adapted from Schmidt RF

ered and the basic patterns are released to function reflexly. Diagnostically these patterns are the flexion patterns of the upper extremity in which the shoulder exhibits scapular depression and downward rotation; the arm, adduction and internal rotation; the elbow, flexion; the forearm, pronation of the wrist and flexion of the fingers (Fig. 9–6). The objective of treatment is to release the uninhibited flexion pattern and to retrain the overwhelmed and paralytic agonists, that is, scapular elevation and retraction, arm abduction and external rotation, elbow extension, forearm supination, and wrist-finger extension.

The spastic stage may proceed to the synergy stage.

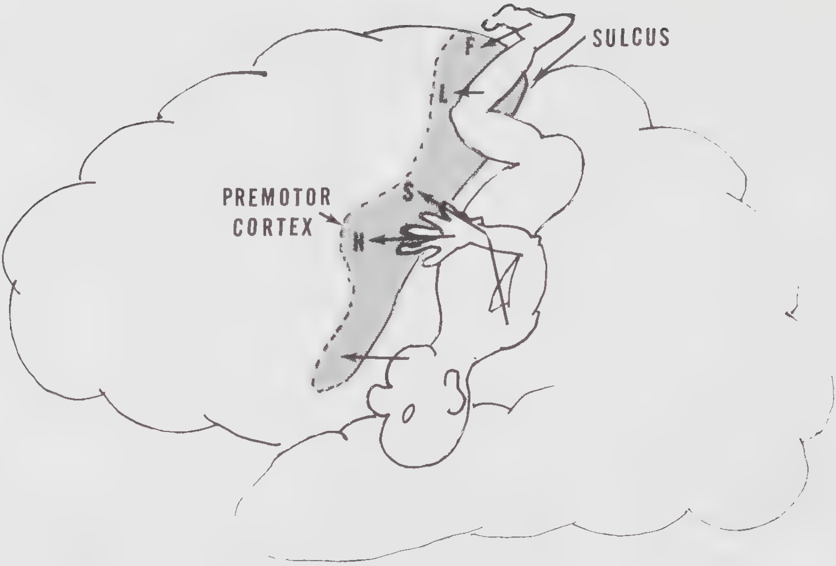


Figure 9–5. Premotor cortex (schematic).

Synergy Stage

The synergy stage is progression of the completed stroke to more severe neurologic impairment in which any effort, external intrusion, or reflex initiates the *total* synergistic pattern. No specific isolated component of the pattern is elicited with voluntary effort.

The therapeutic attempt here is to diminish the unwanted aspects of the synergy and to stress the wanted components of function. In summary, the synergy is uninhibited adduction, internal rotation of the humerus; flexion of the elbow; pronation of the forearm; flexion of the wrist, finger, and thumb. The scapula is externally rotated, protracted, and depressed. The exact motions are overwhelmed. The pattern (synergy) must therapeutically be suppressed, and the opposite movements must be initiated and, it is hoped, regained to normal voluntary function.

THE SHOULDER IN ALL STAGES OF HEMIPLEGIA

The shoulder normally is maintained within the glenoid fossa by (1) the tone of the supraspinatus muscle; (2) the angle of the glenoid fossa maintained facing forward, upward, and outward (Fig. 9–7); and (3) the tone of the scapular

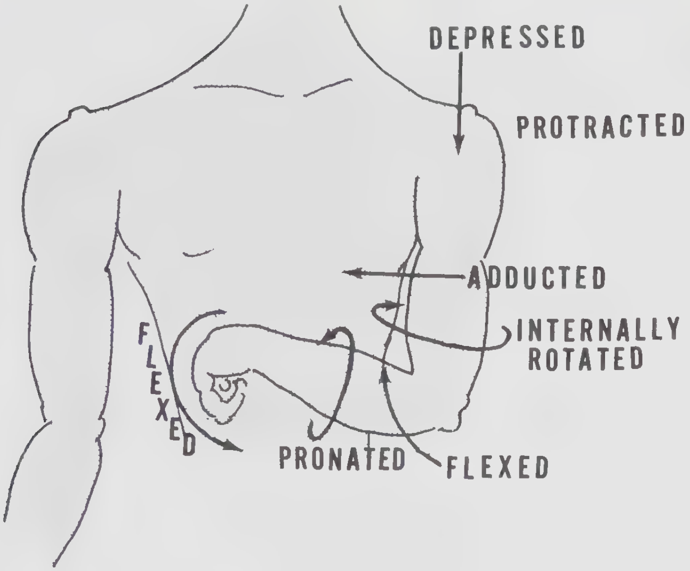

DEPRESSED
PROTRACTED
ADDUCTED
INTERNALLY ROTATED
FLEXED
PRONATED
FLEXED

Figure 9–6. Flexor synergy of the upper extremity.

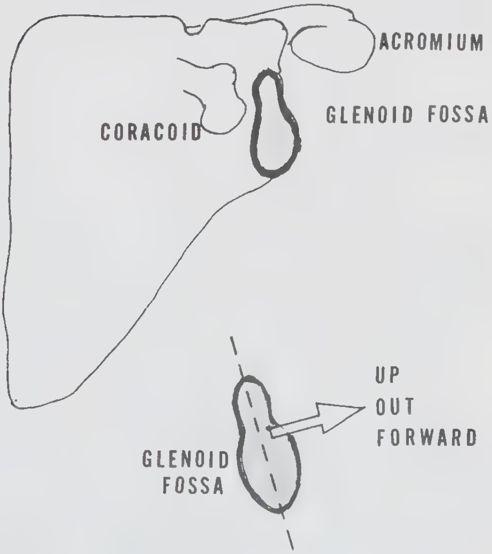

ACROMIUM
GLENOID FOSSA
CORACOID
UP
OUT
FORWARD
GLENOID FOSSA

Figure 9–7. Glenoid fossa. The scapula, viewed from the front, depicts the surface of the glenoid fossa facing forward, upward, and outward.

muscles, which maintain the scapula in its proper alignment with the vertebral column.

The supraspinatus muscle maintains its tone to prevent downward and outward motion of the humeral head by way of the extrafusal fibers of the muscle, which are, in turn, controlled by the spindle and Golgi systems (Fig. 9–8).

The humerus is also partially supported within the glenoid fossa by the superior aspect of the capsule, but this is considered minor because the capsule has insufficient tone to sustain the weight of the dependent arm.

The deltoid muscle also supports the humerus within the fossa by virtue of its intrinsic spindle system and its alignment with the humerus; that is, its origin

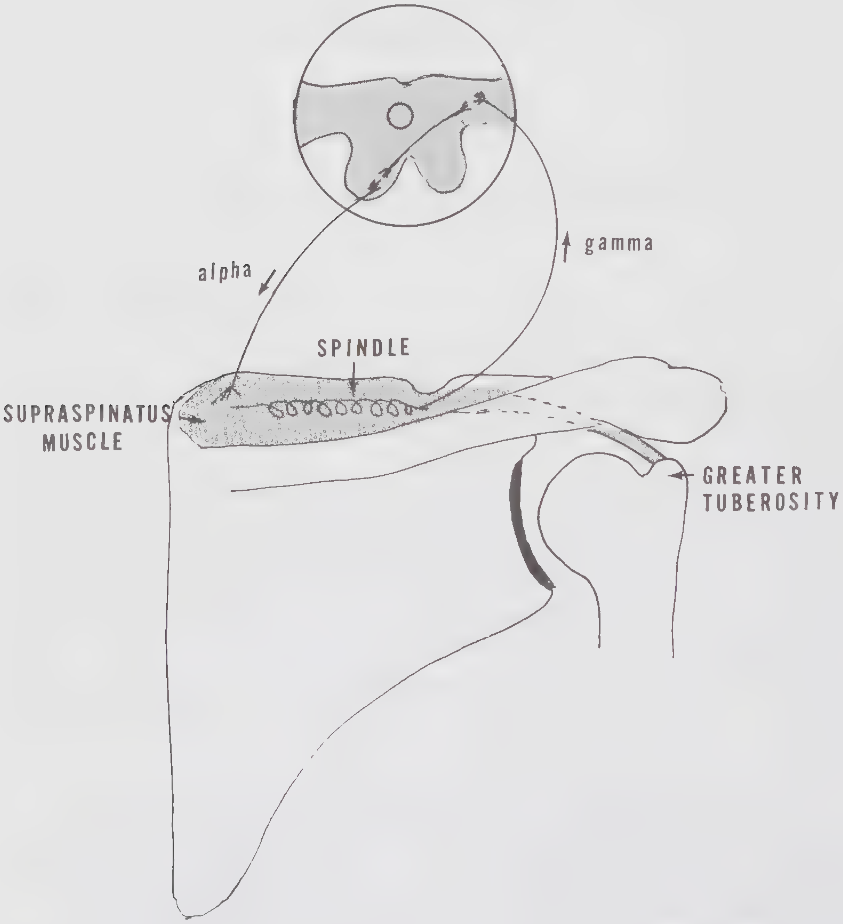


Figure 9–8. Spindle system control of the supraspinatus muscle.

from the acromium and its insertion into the upper humeral shaft.

The scapula is also maintained in its proper alignment with the vertebral column by the tone of the trapezius and serratus anterior muscles. These two scapular muscles also have their own spindle-Golgi systems of innervation.

The vertebral column is also maintained in its erect alignment by the tonus of the erector spinae muscles and *righting reflexes* within the central nervous system.

Any of these neurologic components of the central nervous system—or all of them—that maintain proper glenohumeral shoulder complex position and function can be disrupted in the various stages of the completed stroke.

PATHOMECHANICS OF SHOULDER FUNCTION IN STROKE STAGES

Flaccid Stage

In the flaccid stage there is loss of muscle tone, of voluntary muscular action, and of reflexes. The muscular support of the upper extremity against gravity in the passive phase and muscular contraction in the kinetic phase are lost.

The supraspinatus muscle loses its tone and the humeral head is no longer supported against the overhanging acromium and the coracoacromial ligament. The humeral head thus *subluxes* downward and outward. Only the superior aspect of the glenohumeral joint capsule remains to support the humerus.

The scapula rotates downward and forward due to loss of tone of the trapezius and serratus anterior muscles. This changes the angle of the glenoid fossa, which no longer faces forward, outward, and laterally. In this position the humerus is passively abducted, rendering the supraspinatus muscle less effective. This direction of the plane of the glenoid fossa further allows the head of the humerus to glide downward and outward.

No longer supported by the righting reflexes of the vertebral musculature, the vertical spine bends laterally (Fig. 9–9). The scapulothoracic relationship is altered, furthering the downward and outward rotation of the scapula.

The exact neural mechanism by which the spine bends laterally *toward* the hemiparetic side, implying that it is the lateral erector spinae muscles of the contralateral side that become flaccid rather than those on the hemiparetic side, remains undetermined. It may be that the righting reflexes of the erect spine become impaired. Regardless of the identity of the neural mechanism, it is evident that the spine flexes laterally toward the hemiparetic side.

The combination of all these actions superimposed on a now nonfunctioning spindle-Golgi system, with loss of tone of all the scapulohumeral muscles, causes downward outward subluxation of the glenohumeral joint and excessive stress on the capsule (Fig. 9–10).

The capsule is thin and composed of two tissue layers: an outer layer of

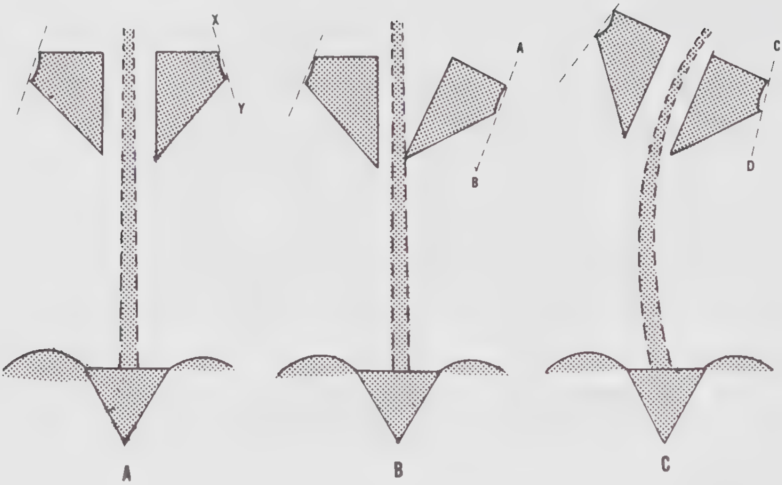


Figure 9–9. Scapular depression. *(A)* Scapular alignment with a straight spine (X–Y glenoid angle). *(B)* Paresis with downward rotation of the scapula (A–B glenoid angle). *(C)* Relative downward rotation of the scapula with functional scoliosis (C–D glenoid angle).

dense fibrous tissue, called *stratum fibrosum*, and an inner synovial layer, called *stratum synovium*. The outer layer is attached to the periosteum by *Sharpey's fibers*. It is poorly vascularized but richly innervated (Hettinga).

The inner layer is highly vascularized but poorly innervated. It is insensitive to pain but highly reactive to heat and cold. It produces hyaluronic acid, which acts as a joint lubricant.

In the flaccid shoulder the added traction upon the joint capsule can well be a source of pain. Traction on the stretched supraspinatus tendon may also be a source of pain, but the joint capsule and the supraspinatus tendon as nociceptor sites have not been confirmed.

Care of the acute flaccid stage of the hemiplegic shoulder must be executed with all these factors in mind. Support of the flail arm mandates correcting and supporting the laterally flexing spine as well as elevating and retracting the scapula. Care must be taken to prevent further passive and active traction on the glenohumeral joint, which can cause further damage to the remaining tissues supporting the joint, until there is return of tone to the supraspinatus muscle. Further discussion of treatment will follow.

Spastic Stage

There may be complete or partial recovery at this stage, or there may be progression to the spastic stage. The total spastic aspect of the upper extremity has

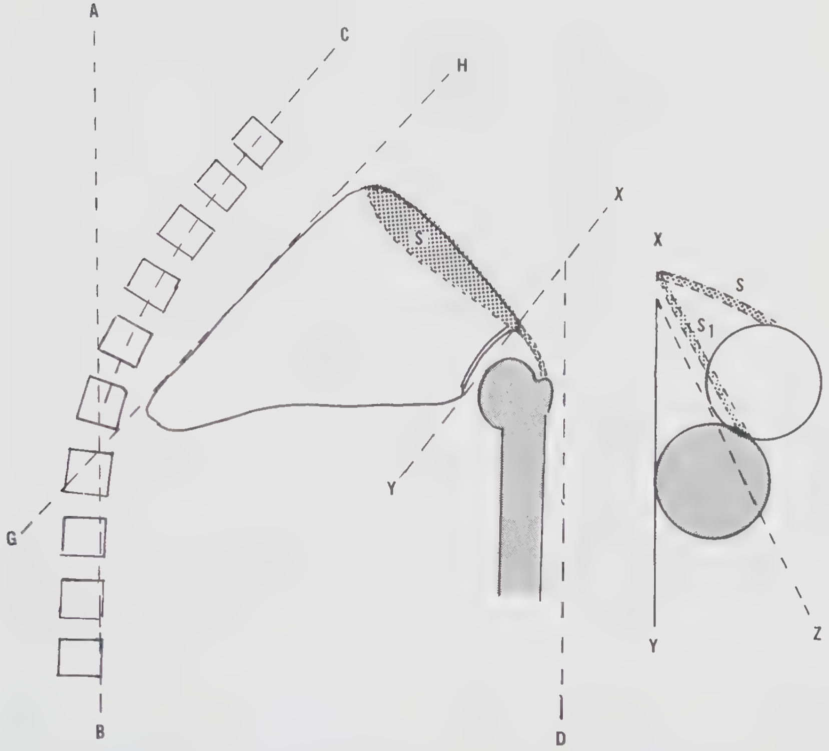


Figure 9–10. Mechanism of glenohumeral subluxation. *(Left)* The lateral curving of the spine (C–B) from erect posture (A–B). The scapula rotates laterally from parallel with the spine to G–H angulation. The alignment of the glenoid fossa (X–Y) becomes erect from its normal upward, outward, lateral facing (X–Z). *(Right)* The tension on the normally aligned glenohumeral joint X–Z with the supraspinatus muscle tendon (S) holds the head of the humerus well-seated. With downward rotation of the scapula, the face of the glenoid fossa becomes vertical (X–Y), and the head of the humerus *rolls* downward, stretching the supraspinatus tendon (S_1).

been well documented (see Fig. 9–6), with the scapula depressed and retracted, the glenohumeral joint subluxed, the humerus adducted and internally rotated.

Spasticity may exist in all or any of these components and with varying degrees of intensity. Spasticity of the muscles of the glenohumeral joint causing internal rotation implies that excessive contracture of the subscapularis muscle is overwhelming the external rotators, that is, supraspinatus, infraspinatus, and teres minor muscles. The agonist-antagonist relationship of the rotators is thus impaired.

There are other muscles that become involved in the spastic internal rotation (Fig. 9–11). The pectoralis major, pectoralis minor, and the latissimus dorsi muscles all play a part, albeit a more minor one than that of the subscapularis muscle.

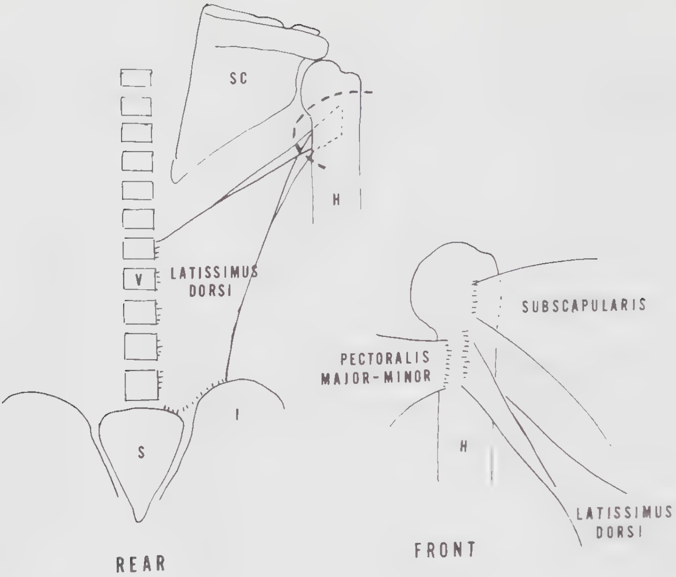


Figure 9–11. Internal rotators of the humerus. The rear figure depicts the latissimus dorsi muscle, which internally rotates and posteriorly flexes the humerus (H) and depresses the scapula (SC).

The front view shows the attachment of the pectoralis major and minor, the subscapularis, and the latissimus dorsi muscles upon the humerus (H). The origin of the latissimus dorsi is the vertebral spine T-5 to L-5 (V), the sacrum (S), and the iliac crests (I).

The muscles of the scapula that cause the shoulder blade to rotate downward and outward are the rhomboids (Fig. 9–12), overwhelming the antagonistic trapezius and serratus anterior muscles. The paraspinous muscles also become spastic, causing further lateral bending of the spine toward the spastic side (see Fig. 9–11). The ipsilateral latissimus dorsi muscle, which attaches anterolaterally to the head of the humerus, also depresses the scapula as well as posteriorly flexing and internally rotating the humerus (see Fig. 9–6).

The spastic phase may be complete, initiating a total synergy, or it may be *partial*, or *incomplete*. Partial or incomplete synergy indicates that the shoulder may become spastic and that the elbow and hand remain flaccid. The opposite may also occur—the hand may become spastic and the shoulder flaccid—as can any combination. With only partial spasticity there may also be degrees of spasticity so that although muscle groups are neurologically spastic they may retain some degree of voluntary control.

Brunnstrom states that, although there may be some variations in the syn-

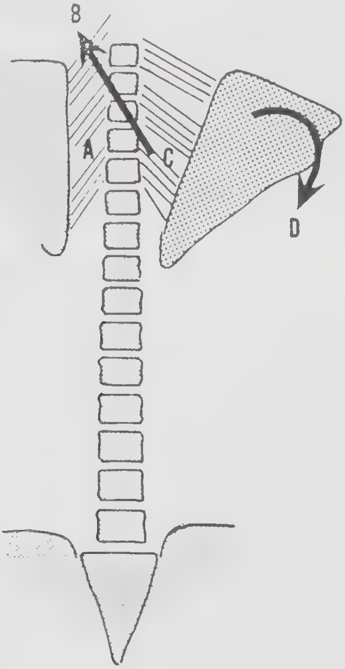


Figure 9–12. Spastic medial scapular muscles. In spasm of the rhomboid muscles, which normally rotate the scapula, the glenoid fossa alignment is lowered and the angulation is made vertical.

ergies, the synergy remains (neurologically) total, the variations being merely the strength of the synergy components.

The initial spastic evolution of the completed stroke from the flail stage also does not remain static. Some progression and/or regression may occur, so frequent evaluation of the hemiparetic extremities must be recorded.

Synergy Stage

This arbitrary stage implies merely that the *entire* synergistic pattern occurs with any reflex action *to* the patient or any motor attempt *by* the patient. The synergy pattern of the upper extremity has been stated to be adduction-internal rotation of the humerus, flexion of the elbow, usually pronation of the forearm—although a few cases present supination—wrist ulnar flexion, finger flexion, and thumb-in-hand position.

SENSORY LOSS IN STROKE

There may be sensory deficit, such as appreciation of touch, vibration, pain, temperature, and possibly position sense. It has been claimed that loss of any or all of these sensory modalities occurs in 80 percent of stroke patients (Van Buskirk and Webster).

In many cases perceptual dysfunction may also occur. In addition to loss of spatial relationship, there may also be loss of body image, neglect, or denial. When there is a severe or significant perceptual deficit the prognosis for recovery is adversely influenced.

FUNCTIONAL RECOVERY

There is some spontaneous recovery of voluntary function in the patient with a completed stroke that needs consideration before there is any discussion of the pathomechanics of the hemiplegic shoulder and a discussion of treatment.

There has been a paucity of controlled studies regarding functional recovery, which makes it difficult to evaluate the efficacy of treatment protocols. Van Buskirk claimed that spontaneous recovery occurred chiefly in the 2 to 3 months following the stroke.

The sequence of recovery was described in Twitchell's classic paper. First voluntary motion occurred within 6 to 33 days after cerebral vascular accident. The first motion was noted in the shoulder: slight flexion. The remainder of recovery occurred in the arm, with flexion of the elbow, wrist, then the fingers. The initial motion was essentially a part of the flexor synergy. Twitchell claimed that the flexor synergy was followed by some extensor patterns.

The synergy patterns, at first merely reflexive, were followed by increasing control of the synergies until voluntary control improved with ultimate individual joint motion control. These, Twitchell affirmed, were *recovery* patterns when there was recovery.

Bard and Hirschberg's frequently mentioned paper claimed that 40 percent of their series recovered "full motion of the involved upper extremity." Whether this recovered motion implied also full functional recovery is not ascertained. Their conclusion was that in patients who recovered full motion, there was initial return of *voluntary* function within the first 2 weeks. They also found that when recovery began within the first 2 weeks, recovery was equal in proximal and distal musculature. Of the remaining stroke patients, 40 percent had partial recovery, and 20 percent experienced no recovery.

In his studies, Carroll found that if there was no return of motor function within the first week, it was improbable that full recovery of the upper extremity would occur. Carroll indicated *functional* recovery as opposed to motor recovery, a more meaningful designation.

Two more recent papers (Carroll, Shah and Corones) evaluated 100 patients in regard to functional recovery. They found that 34 percent recovered grasp of the hand but no repetitive finger activities. Only approximately 24 percent could turn a door knob, and they regained little or no individual finger function. Even in these cases, the position of the hand and where it is placed would be of limited practical value.

Bach-y-Rita has written probably the most extensive literature on brain plasticity as the basis for recovery of function. It is his claim that prolonged treatment over the course of many months to many years has functional benefit, a contradiction of the prognostic studies mentioned above.

TREATMENT CONCEPTS OF THE HEMIPLEGIC SHOULDER

Flaccid Stage

Every aspect of the flail shoulder must be considered: the thoracic spine, the scapula, and the glenohumeral joint. Much of the failure to regain function and to avoid or to minimize pain can be attributed to failing to consider *all* the components of the shoulder complex.

It must also be accepted that the sensory components as well as the motor components of the impaired shoulder must be addressed. Considering the position of the flail extremity and its relationship to gravity and total body position, proper positioning must be used to neutralize undesirable and injurious positions of the upper extremity.

1. The supine position must be avoided, but when it is assumed; the arm must be positioned to elevate the humerus into the glenoid area and the scapula must be protracted.
2. The patient must be assisted to lie on the side rather than to lie supine. The shoulder should be slightly forward flexed and the elbow extended. This positional change has been assisted by an air-inflated sleeve (Johnstone) (Fig. 9–13), which permits concentration on the shoulder while ignoring the remainder of the upper extremity. Prolonged use of this sleeve, however, is not advised, inasmuch as it also maintains the fingers at full extension and denies *pump* action of the wrist and fingers.
3. Passive transfer of the patient must be performed by holding the scapula rather than by traction on the arm.
4. The patient's head should be laterally flexed and rotated toward the unaffected side.
5. In the seated position the trunk must be supported in the erect position and the arm supported upward into the scapula by the use of a table board and pillows.
6. The trunk should be supported laterally flexed away from the affected side.
7. Unrelated directly to the shoulder, the hand and fingers should be supported in a wrist-extended and finger-flexed position.

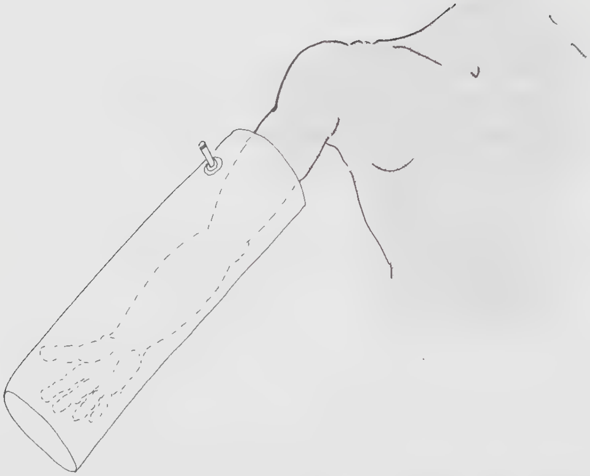


Figure 9–13. The entire upper extremity is enclosed within an air-inflated splint. The warmth of the breath that inflates the splint and the complete skin contact supposedly act as reflex stimulators. The elbow is extended, as are the wrist and fingers, and the entire arm can be placed in abduction and external rotation. Thus the shoulder can be positioned and exercised. (Johnstone air sleeve.)

Contrary to the generally held concept that pressure upon the affected arm must be avoided, it is permissible—and desirable—to have the patient lie on the affected side, provided that the scapula is protracted (placed forward). In that position pressure is not directly upon the glenohumeral joint.

Before any active rehabilitation exercises are begun for the extremities, trunk motion must be initiated. Rolling from side to side must be started, but throughout this trunk rolling, certain positions of the upper extremity should be avoided:

1. Retraction (posterior flexion) of the shoulder
2. Depression of the scapula
3. Adduction of the arm
4. Internal rotation of the arm
5. Elbow flexion
6. Pronation of the forearm
7. Ulnar deviation of the wrist
8. Flexion of the wrist
9. Adduction of the thumb

As the patient gradually learns to move from the supine position to the prone position, the arm should be flexed forward at the shoulder with the elbow extended. In the supine position the arm should be supinated at the forearm, extended at the wrist and elbow with the thumb abducted. The arm should gradually be raised overhead, maintaining the arm fully extended at the elbow and wrist and the entire upper extremity externally rotated (Fig. 9–14).

Inasmuch as there is no tone in the flaccid stage, contracture of the shoulder joint need not concern the therapist. Full range of motion needs to be evaluated often, because the completed stroke progresses or regresses almost daily. Full range of motion, however, need not be a therapeutic indication—merely a frequent evaluation.

Throughout all passive movements of the upper extremity (Fig. 9–15) the patient should be asked to *try* to assist in motion or *holding* in all the acquired positions. This initiates and encourages ultimate active control of the extremity.

Sensory stimulation to the extremity is also indicated to initiate ultimate active motion. This can be brushing, stroking, tapping, or even gentle stretch. Electrical stimulation of the shoulder muscles has its advocates in reestablishing active muscular control.

When the patient begins regaining the sitting position, the sound arm is invoked but the impaired arm must be specifically addressed. Gradual gentle leaning on the extended arm (Fig. 9–16) reduces the glenohumeral subluxation and initiates proprioceptive stimulation to the musculature of the shoulder. The flail elbow may need support. The wrist should be placed in an extended position with the arm slightly externally rotated.

In the seated position the arm can be elevated into the glenohumeral joint by proper use of the tray table and pillows while the patient simultaneously *leans* on the afflicted arm.

SUBLUXATION OF THE SHOULDER

One of the most perplexing complications of the hemiplegic shoulder is subluxation of the glenohumeral joint. Subluxation allegedly initially occurs during the flaccid stage (as noted in the earlier portions of this chapter) and often persists into the spastic stages and even into the recovered completed stroke.

Pain in the hemiplegic shoulder is considered by many clinicians to be the result of subluxation by virtue of traction on the capsule and the rotator cuff muscles. The exact mechanism of pain has yet to be documented, inasmuch as there are many cases of subluxation of varying degrees with *no* pain and many painful shoulders with no significant subluxation.

Treatment to prevent subluxation occupies much of therapy time, and it is in this aspect of therapy of hemiplegia that much controversy exists.

Authorities in the field of rehabilitation, considered subluxation as a cause of pain, disability, and ultimately frozen shoulder (Bierman and Licht, 1952). This was corroborated by Tobis, who attributed the pain to capsular and cuff muscle stretch. This concept has recently been questioned, and many now ignore subluxation, per se, other than to avoid excessive traction on the joint.

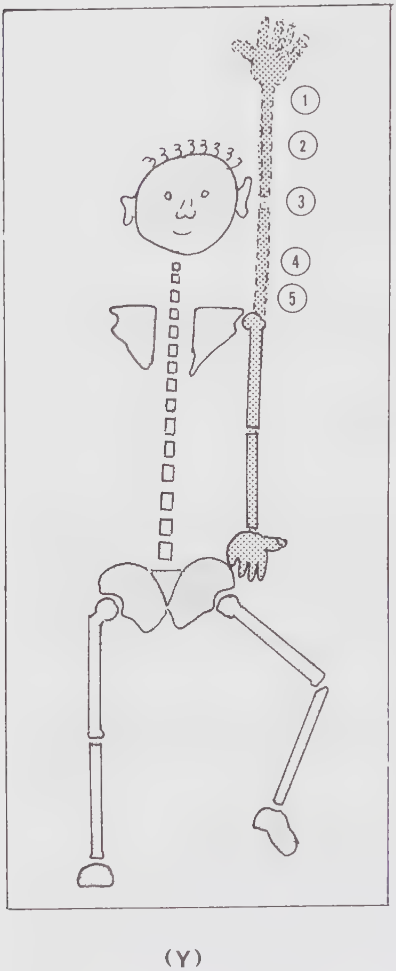


Figure 9–14. With patient supine, the extremity is gradually raised to the overhead position: 1 and 2, the forearm is supinated; 3, the elbow extended; 4, abducted; 5, the upper arm externally rotated.

Diagnosis of subluxation remains a clinical observation; there is no accurate measurement to quantify the degree of subluxation. An accurate measurement of subluxation would validate the benefit, or lack thereof, of treatment addressing subluxation, but none to date has been standardized.

The difficulty in such a measurement is evident when one considers all the aspects of glenohumeral subluxation:

1. Separation of the humeral head from the overhanging acromium
2. Lateral separation of the humeral head from the glenoid fossa
3. Change in the angulation of the scapula from the vertebral column
4. Separation of the scapula medial margin from the thoracic spine
5. Lateral angulation (curving) of the thoracic spine
6. Degree of dorsal kyphosis

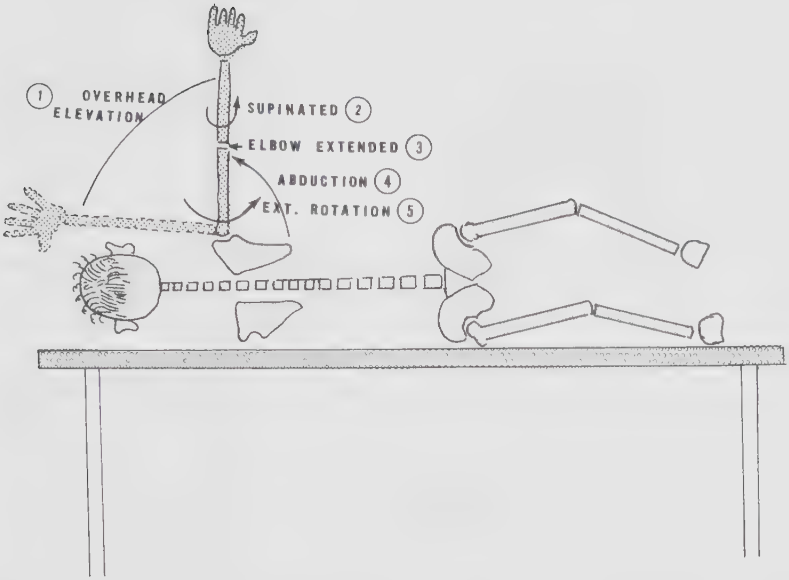


Figure 9–15. In passive exercise, the patient tries to hold the extremity in various positions (1–5).

Treatment of subluxation by the use of a sling remains controversial, but the literature remains replete with numerous medical articles. Several are illustrated here (Fig. 9–17, 9–18, 9–19), but none can be advocated as beneficial in preventing or diminishing the degree of subluxation; preventing pain from subluxation, or preventing complications during the ultimate stages of progression or recovery.

Hurd and colleagues, among many authors, concluded that the commonly used hemisling had no appreciable effect on the ultimate range of motion, subluxation, pain, or peripheral traction injury. Many actually implicate the sling as aggravating further subluxation, intensifying the extent of subsequent synergy, and some claim that a sling position of the hemiplegic arm impairs proprioception and is thus detrimental to proper gait.

The Rood splint (see Fig. 9–15), initially designed to afford proprioceptive stimulation of the glenohumeral joint capsule, actually causes further lateral subluxation and is now largely discredited.

A wheelchair support (Fig. 9–20) was initially designed to minimize edema of the arm while decreasing the degree of pain and subluxation. If properly supervised and supplemented with other, more active, therapy, this sling can be indicated and beneficial.

Recent literature has implicated significant impairment of the hemiplegic shoulder from traction injury to the brachial plexus. Undoubtedly this can and

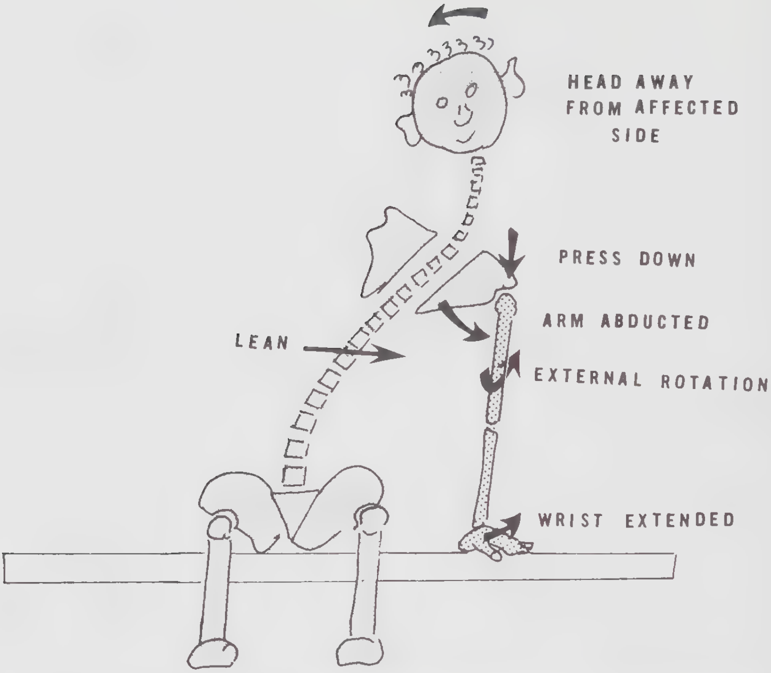


Figure 9–16. When the patient sits, he or she should support his or her weight on the affected side in the manner depicted.

does occur, but I find that it is far less frequent than the literature implies. It should always be considered in the differential diagnosis when there appears to be more or different neurologic impairments than would be expected. The condition of brachial plexus injury has been fully considered in the previous chapter on the neurologic aspects of shoulder pain and impairment.

THE HEMIPLEGIC SHOULDER IN THE SPASTIC STAGE

The flaccid stage usually evolves into the spastic stage in the completed stroke patient. Spasticity comes insidiously, predominantly involving the flexors of the upper extremity and the extensors of the lower extremity.

In the upper extremity the shoulder depressors and the fixators of the scapula are involved early (rhomboids) (see Fig. 9–12). The latissimus dorsi also undergoes spasticity, bringing the scapula downward and rotated as well as adducting, posteriorly flexing, and internally rotating the humerus.

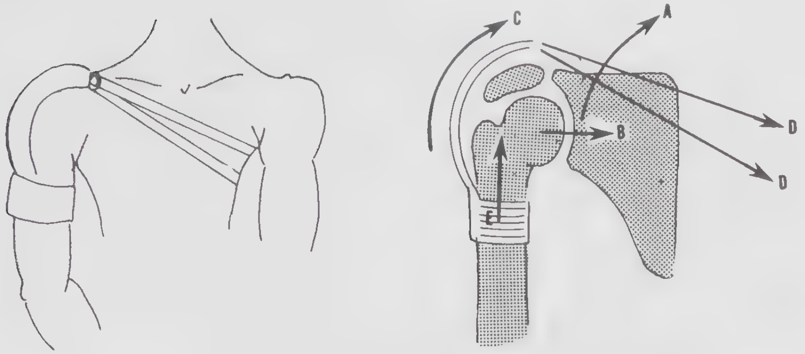


Figure 9–17. Proposed design for prevention of subluxation. The glenohumeral joint is elevated to the desired physiologic angulation (A). The head of the humerus is adducted into the *seated* position (B). The humerus is elevated into the suprahumeral fossa (C). The cuff is replaced by the splint (D). The sling attaches to the humerus at the site of deltoid insertion and elevates the humerus into the suprahumeral joint space (E). (From Cailliet, p 69, with permission.)

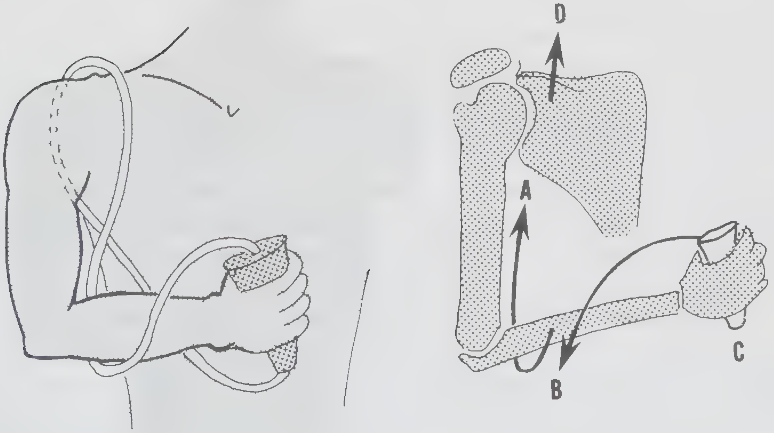


Figure 9–18. Rood sling. Being elastic tubing, the support gives kinetic support (A) and stimulates extension of the arm. By proper application, the forearm is supinated (B). The hand holds a cone, (C) which spreads the fingers and thumb while radially deviating the wrist. The scapula is elevated and derotated (D). (From Cailliet, page 69, with permission.)

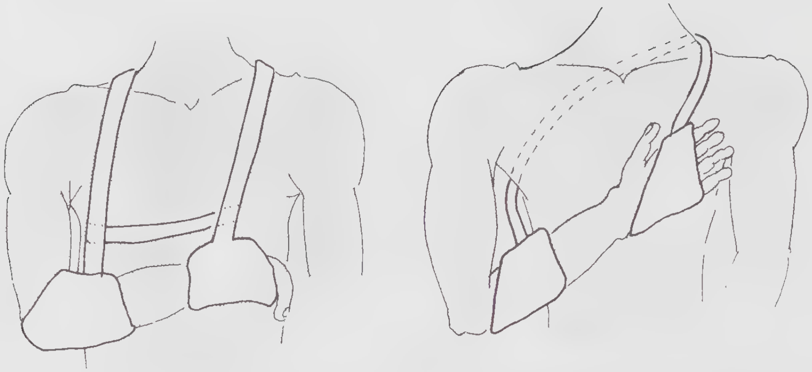


Figure 9–19. Shoulder slings. These slings are designed to support the arm and to minimize downward subluxation of the glenohumeral joint. They have not been proven to prevent subluxation or to hold the arm in a flex position. (From Cailliet, page 68, with permission.)

In the flexor synergy the pectoralis major and pectoralis minor muscles also become involved as especially does the subscapularis muscle. The biceps muscle is also involved within the synergy, and the biceps tendon, angling toward the upper border of the glenoid fossa through the bicipital groove of the humerus, tends to depress the head of the humerus.

With severe neurologic deficit the neck reflexes become released from voluntary upper cortical control and exert action on the upper extremity. The arm may extend at the elbow with the head turned toward that side and flex at the elbow when the head is turned away from the involved side. The arms may assume an extended position when the head-neck is extended (Fig. 9–21), because there may be a flexion pattern when the head-neck are flexed. These are the classic *tonic neck reflexes* that will frequently be elicited but must be tested to see if they are operational in each individual patient.

Their presence indicates a severity of neurologic deficit, and although they are frequently utilized in therapy (Knott and Voss), their presence can be considered to be prognostically unfavorable for ultimate recovery.

Just as there is gradual evolution of the completed stroke from flaccid to spastic stages, there are varying degrees of involvement and severity. Not all aspects of the synergy evolve to the same degree, and not all aspects of the synergy are predictable. For example, the hand-wrist and fingers synergy may differ when the forearm is pronated rather than supinated. The shoulder synergy pattern remains reasonably consistent.

With further loss of cortical control incurred from the stroke, the synergic patterns emerge:

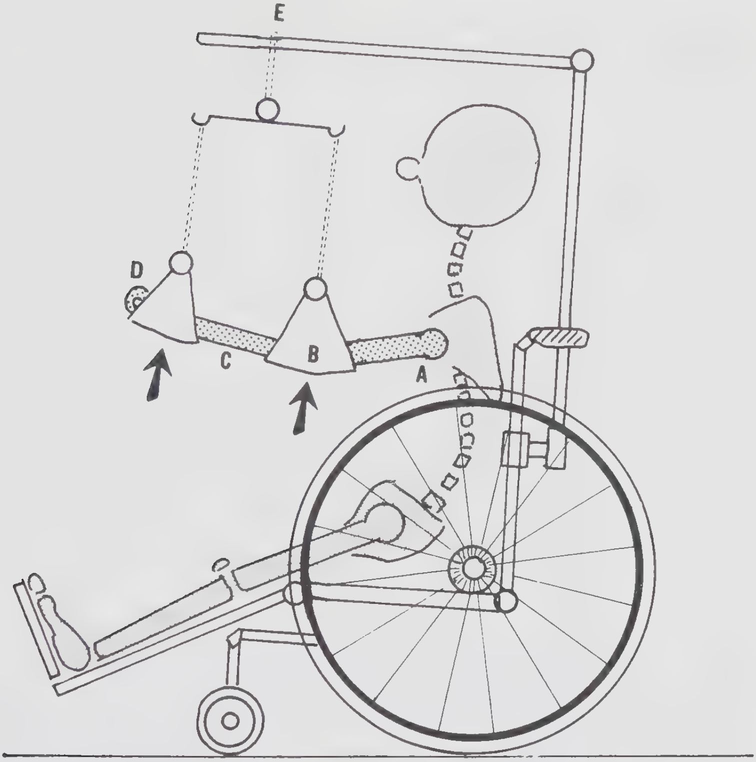


Figure 9–20. Wheelchair arm sling. The shoulder is held forward, flexed, and adducted (A). The elbow is supported in an extended position (B). The wrist and fingers are held extended (C, D). Movement of the entire arm is permitted (E). (From Cailliet, page 70, with permission.)

1. Upper arm adducted and rotated internally
2. Scapula retracted and rotated downward
3. Elbow flexed to an acute angle
4. Forearm pronated (occasionally supinated)

Jackson described these basic neurophysiologic patterns of the middle motor center (see Figs. 9–3, 9–4, 9–5) as differing from the extensor synergy, in which there is

1. Protraction of the scapula
2. Forward flexion of the arm toward the front of the body
3. Internal rotation of the arm
4. Extension of the elbow
5. Pronation of the forearm

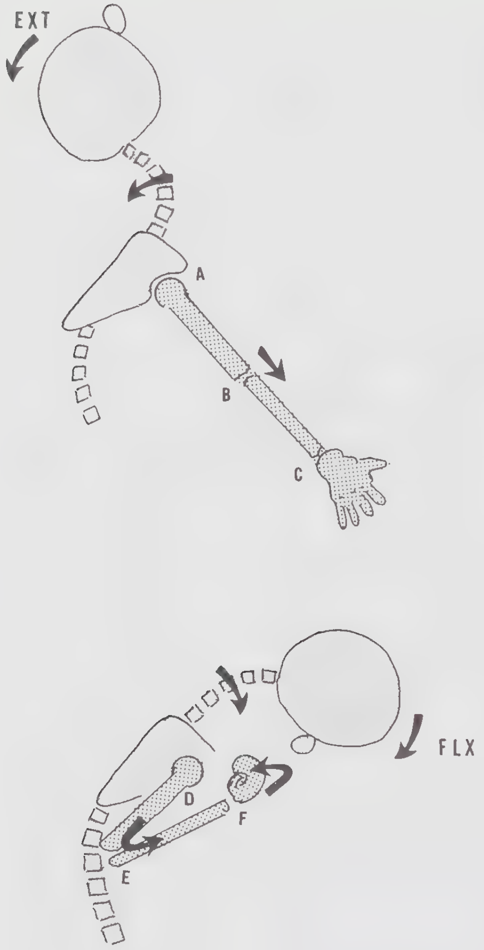


Figure 9–21. Upper illustration depicts the extensor pattern of the upper extremity in neck extension. The lower drawing shows flexion of the upper extremity on neck flexion. A, shoulder; B, elbow; C, wrist; E, elbow flexed; F, wrist-fingers flexed. (From Cailliet, page 57, with permission.)

Because the synergy emerges in the completed stroke, any component of the flexor or extensor synergies can evolve. Usually the elbow component occurs first with spastic flexion. The shoulder phase is weaker and may never occur unless the patient progresses to a more reflex status.

When the hemiplegic patient attempts abduction of the upper extremity, only certain movements occur. The scapula elevates with little if any abduction of the upper arm at the glenohumeral joint. This can be considered a *shrugging mechanism,* similar to that noted in acute supraspinatus tendinitis.

The spastic scapular adductors may prevent the scapula from moving forward when there is attempt to flex the upper arm forward. Persistence of this scapular spasticity presents a therapeutic challenge if there is to be any progression of meaningful function of the upper extremity.

The strongest component of the scapular spasticity appears to be the pectoralis major. It is in this muscle that the first evidence of upper extremity spasticity is noted. Next the forearm pronators undergo significant spasticity.

Because significant internal rotator spasticity is noted early, it is apparent that spasticity exists in the subscapularis muscle as well as in the pectoralis major.

The tonic neck reflexes have been mentioned but the trunk movement also has a profound effect on movements of the upper extremity. Rotation of the upper trunk on the lower trunk facilitates or inhibits reflex patterns as well as voluntary patterns, and it must be evaluated clinically.

Hemiplegic patients frequently exhibit associated pattern reactions when motion is attempted on the contralateral *normal* side. There are no consistent reactive patterns, thus the clinical significance of such a contralateral reflex activity must be decided when it is elicited.

The sensory involvement of the upper extremity must also be evaluated in assessing functional loss and impairment. In addition to loss of touch, vibration, and position sense, there may be denial (Fig. 9–22), or *astereognosis,* in which the patient fails to recognize familiar objects such as coins, keys, pins, pens, and so forth.

As voluntary control returns in spontaneous recovery, portions of the synergy return. Every component assumes attention in the therapeutic regimen in order to enforce the return of the remaining, still uncontrolled, synergy components.

All movements normally are responses to sensory stimuli, which act through exteroceptors, vision, touch, and hearing (see Fig. 9–4). The proprioceptors send their messages from the muscle spindles, tendon Golgi apparatus, joint and tendon proprioceptors, skin receptors, and visual impressions. This sensory modality is instrumental in all treatment protocols.

The Bobath concept is that the problem of hemiplegia lies in the failure of the patient to control isolated voluntary components of the synergy patterns. Bobath techniques intend to change "abnormal patterns of movement," contrary to the proprioceptive neuromuscular facilitation techniques involving heavy resistance, which are advocated by Knott and Waters. They also differ from Brunnstrom's use of *associated patterns*, which the Bobaths feel should be avoided.

The Bobath approach is based on the belief that the synergy reflexes are *reinforced* by these other techniques rather than avoided and controlled. The Bobaths' concept of *voluntary muscular weakness* is as follows:

1. The weakness is due to being relatively overwhelmed by the antagonist spastic muscles.
2. The prime mover muscle(s) may be weak in voluntary motion but *strong* in abnormal synergy motor patterns.
3. Weakness may be caused by sensory deficit(s).
4. Soft tissue periarticular contracture can enhance or simulate weakness.

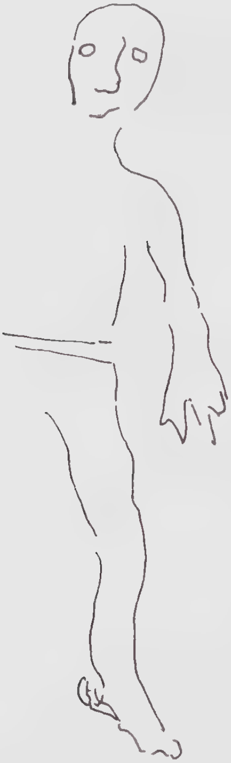


Figure 9–22. Denial demonstrated by patient. When a patient is asked to draw his body or a person, he is aware of only one half of his body, denying the existence of the other half. This is such a drawing from a patient. (From Cailliet, page 77, with permission.)

The Bobaths feel that the problem of spasticity and release of the synergies must be addressed before voluntary motor activity can be achieved. They attempt to achieve this objective by *special handling* of the patient and by controlling certain aspects of the pattern, which they term *key points* of control. These key points are the proximal neck, spine, shoulder, and pelvis. An important key point in the upper extremity, they claim, is rotation of the shoulder girdle in relationship to the trunk and pelvis. They also emphasize the neck rotation in relationship to the pelvis during shoulder-arm patterns.

Generally, in the hemiplegic patient there is no reciprocal relaxation of the antagonist muscles on contraction of the agonist muscles. There is actually cocontraction; that is, simultaneous contractions of the agonists *and* antagonists. This is the basis for recent introduction of biofeedback in therapy regimens: to relax the antagonists, thus releasing the prime movers to be more effective. Previously antagonists have been *removed* or diminished by denervation techniques, such as phenol nerve blocks, but the results have been unpredictable and followed by painful sequelae.

In the spastic hemiplegic shoulder the muscles that become affected initially are the fixator and depressors of the scapula. Because the depressors are more powerful than the elevators, the scapula becomes depressed and adducted, causing the upper arm to be abducted at the glenohumeral joint yet adducted and internally rotated by the imbalanced rotator cuff muscles. The remainder of the upper extremity synergy follows at the elbow, forearm, wrist, and fingers.

Before reeducating the total upper extremity, it is imperative to release the antagonist spastic muscles of the scapula and scapulohumeral joints and to retrain the opposed agonist muscles.

When the patient begins sitting balance training, the arm must always be held in the proper position and the humerus must exert *upward* pressure against the coracoacromial ligament and the acromium. Downward traction upon the arm may stimulate some motor responses (Knott and Voss), but upward pressure stimulates postural reflexes (Bobath).

The techniques of muscle reeducation cannot be given full consideration and evaluation here. There are numerous texts discussing the theories, principles, and techniques of current hemiplegic therapy, many of which are listed in the references. The basic concepts, however, are enumerated here for general consideration in treating the hemiplegic patient and especially the hemiplegic shoulder:

1. Visualization of specific movement by the patient must be utilized by the therapist; for example, using a mirror.
2. Verbal reinforcement of the intended and attempted motion must be conveyed to the patient.
3. Similar motion of the other (normal) shoulder done simultaneously is instructional and reinforcing.
4. Encouraging the *feel* of a specific motion by the patient reinforces ultimate voluntary motion.
5. Decrease the depression and retraction of the scapula by numerous positions of the entire upper extremity.
6. Simultaneously apply sensory stimulation by tapping, stroking, rubbing, and scratching the skin over the portion of the extremity being addressed.
7. Initiate prone exercises as well as sitting and standing exercises, such as kneeling, crawling, and turning over from prone to supine (Fig. 9–23).

These prone exercises stimulate righting reflexes as well as imitating primitive motor functions.

While in the seated position stimulation exercises and positions can be initiated that will promote sensory stimulation and decrease subluxation of the shoulder as well as begin modification of the undesirable synergy patterns (Fig. 9–24).

8. When relaxing the spastic antagonist muscle groups, consider the following:

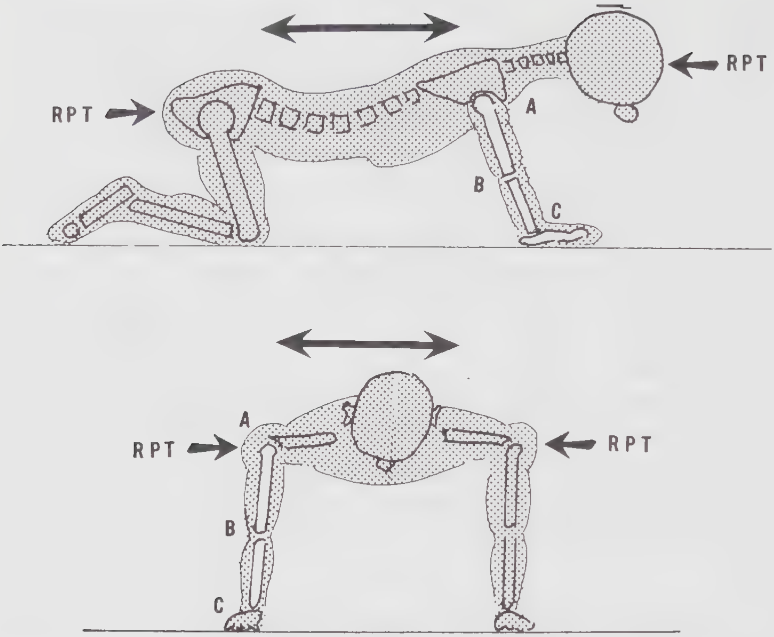


Figure 9–23. Patient crawling with weight on affected arm. RPT indicates resisted pressure from therapist. (From Cailliet, p 81, with permission.)

a. Increase passive range of motion, but avoid stimulation of the stretch reflex. This is accomplished by gentle slow motion. It may also be enhanced by simultaneous use of an ice pack to the involved articulation.

b. Contraction of the spastic antagonist muscle group (if under some degree of voluntary control) should be followed by relaxation. This is a principle of biofeedback. It can also be followed immediately by slow gentle stretching of the antagonist muscle group.

c. When there is some evidence of voluntary control of either agonist or antagonist muscle groups alternate contraction of each group. This is termed *rhythmic stabilization* and is advocated by Knott and Voss in their proprioception neuromuscular facilitation techniques (Fig. 9–25).

9. Some techniques recommend *vigorous stretching* of the spastic muscle groups to stretch the connective tissues of the joints and tendons and allegedly to decrease muscle spindle activity (Kottke). I question this, but it is worth an attempt in care of the hemiplegic patient if vigorous *traction* is not also initiated.
10. Electrical stimulation has its advocates to enhance contraction of the agonists and the antagonists for ultimate *relaxation*.
11. Bilateral training in which the normal arm is exercised simultaneously

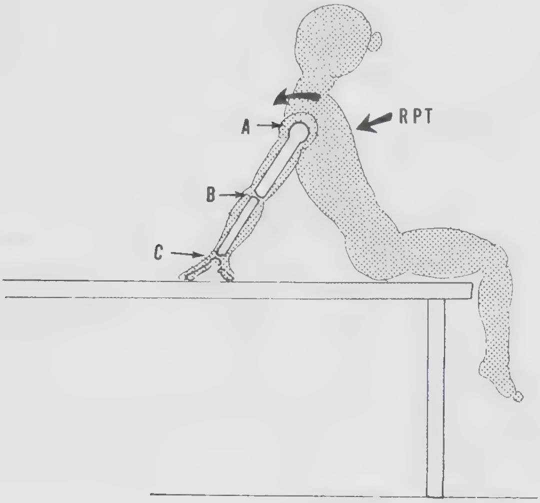


Figure 9–24. Patient sitting and leaning on affected arm. RPT indicates patient resists pressure from therapist. (From Cailliet, p 82, with permission.)

with the paretic arm is effective. The normal arm actually treats the abnormal one and stimulates proprioception as well as helps it regain range of motion.

12. Use of modalities such as ice massage, TENS, vibration, and *trigger point* pressure may diminish spasticity.

Biofeedback has been recently advocated increasingly by research physiologists and clinical therapy centers and merits application and further research. Biofeedback implies muscular relaxation by verbal, visual, sensory, or auditory responses. Electromyographics feedback employs these responses and is becoming a research tool as well as a treatment modality.

Sensory deficits, which are varied and numerous, represent a tremendous obstacle to meaningful functional recovery. Before significant efforts are extended on therapy procedures, these sensory deficits must be ascertained, clarified, and addressed.

Perceptual deficits are especially deleterious to recovery. Brain defined those with perceptual deficits as "people who were guessing the meaning of a language for which they did not know the alphabet."

The following must be investigated in evaluating functional impairment of a hemiparetic patient:

1. Mental status
    a. Oriented as to time, place, and identity

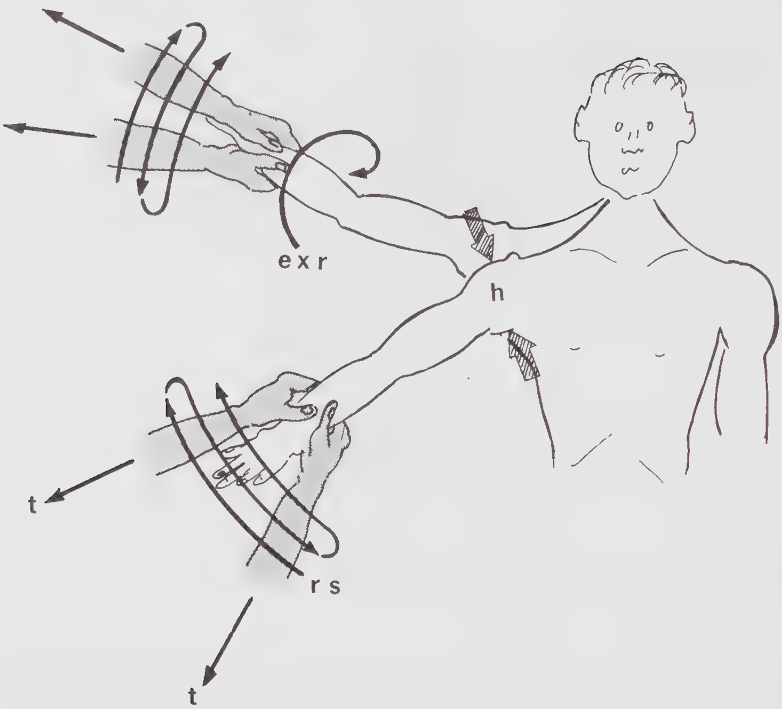


Figure 9–25. Rhythmic stabilization technique to increase shoulder joint range of motion. As the therapist elevates, abducts, and externally rotates (exr) the arm with some traction (t), the patient resists to prevent motion. The exact opposite motion of the arm is then attempted by the therapist and resisted by the patient. The same amount of force and resistance must be exerted by the therapist as by the patient. Minimal joint motion occurs (h), and most muscular contraction is isometric. (From Cailliet, page 83, with permission.)

   b. Memory—especially to recall immediate or short-term events
   c. Attention span: clarifying the ability and duration of ability to concentrate on an activity
2. The presence of apraxia, which may be confused with a perceptual loss
3. A premorbid condition such as a hearing defect, speech impairment, or visual deficit.

There are many unfavorable predictive factors in estimating the functional recovery of any patient. Sensory impairment and perceptional deficit have been mentioned. Impaired peripheral vision is a negative prognostic factor as is excessive profound spasticity and a prolonged flaccid stage.

After a full evaluation of the patient's impairment—functional as well as neurologic—there is no accepted *standard* method or technique of *muscle reeducation.* The term is misleading, because *reeducation* is the regaining or re-training of normal function of neurologic control with the peripheral musculature being merely the end organs of function.

HETEROTROPIC OSSIFICATION

A true ossification process is a complication that occurs in neurologically damaged patients. Essentially more frequently found in spinal cord injuries and brain-damaged patients, it can also complicate the care of the patient with the completed stroke.

There are three forms of tissue ossification noted:

1. Neurogenic heterotropic ossification, found in severely neurologically damaged patients, such as those with spinal cord injuries and severe brain damage; these result from a form of trauma
2. Direct muscular trauma resulting in ossification, often after significant muscle tissue damage and local hemorrhage
3. The congenital myositis ossificans progressiva.

The first of these categories, the neurogenic heterotropic ossificans that is found as a complication in a completed stroke, begins as a soft tissue calcification around a neurologically affected joint. It should not, by rights, be termed a *myositis*, inasmuch as this would indicate inflammation of muscle, nor should it be considered an *arthropathy*, because this term implies articular inflammation, which is not involved. Hence the term *heterotropic ossification* is used, which excludes the other two implications.

The condition was first revealed as a neurologic entity by Riedel in 1883, when it was associated with hemiplegia following a cerebrovascular accident. Numerous other forms of neurologic injuries have since been added to the causative factors.

Heterotropic ossification involves the hips, knees, *shoulders*, and elbows, in that order of frequency. It may be unilateral, but it also may be bilateral and involve other joints simultaneously.

The cause of heterotropic calcification remains unknown. The condition is a true ossification process and microscopically resembles normal bone. All aspects of origin have been summarized by Garrison in a recent article.

Heterotropic ossification is usually symptomless and is incidentally discovered radiologically in a joint that appears to be losing its normal active and passive range of motion. Clinically, there may be a sudden area of erythema, warmth, and local swelling, which may gradually progress in the extremity. When the mass occurs in the vicinity of a joint, there may be a rapid onset of joint limitation.

Onset occurs as early as 2 weeks after the stroke but may occur as late as 3 to 6 months afterward. The x-ray findings may not be noted for 7 to 10 days after the onset of local clinical swelling, rubor, and erythema. Laboratory findings of elevated serum alkaline phosphatase occur before positive x-ray verification.

Usually there are four stages of heterotropic ossification delineated (Nicholas):

Stage 1: Tissue swelling and rubor with an elevated blood alkaline phosphatase. This stage found in a neurologically involved patient alerts the clinician to the possibility of imminent heterotropic ossification.

Stage 2: Tissue swelling, an elevated alkaline phosphatase, and radiologic evidence of beginning ossification.

Stage 3: Minimal if any evidence of tissue inflammation but remaining elevated alkaline phosphatase and radiologic findings.

Stage 4: Merely remaining radiologic ossification. Evidence of inflammation and elevated serum alkaline phosphatase is gone. The findings at biopsy are those of well-organized bone, but all other clinical findings are gone.

The clinical findings during these stages are not parallel regarding symptoms and joint limitation. They essentially document the pathologic changes of the four stages.

Treatment

Because the sequelae of heterotopic ossification are often suspected by a rapidly decreasing range of motion as well as local swelling and rubor in the periarticular tissues, it is mandatory that active physical and occupational therapies be instituted to retain or to regain range of motion. Ultrasound, microwave diathermy, and other forms of heat have been advocated, but there is no verification that these modalities, per se, are significantly effective, other than enhancing the active and passive range of motion and decreasing discomfort.

Oral steroids may decrease inflammation but have no confirmed benefit to the ultimate ossification. Nonsteroidal anti-inflammatory medications also fall in this category of palliative therapy.

Surgical removal of the ossification should be considered only when the bone is considered mature and is clinically ascertained to be inhibitory to expected range of motion.

BIBLIOGRAPHY

Anderson, TP, et al: Predictive factors in stroke rehabilitation. Arch Phys Med Rehabil 55:545, 1974.
Ayres, AJ: Perception of space of adult hemiplegic patients. Arch Phys Med Rehabil 43:552, 1962.
Bach-y-Rita, P: Brain plasticity as a basis of the development of rehabilitation procedures for hemiplegia. Scand J Rehabil Med 13:73, 1981.
Bard, G and Hirschberg, GG: Recovery of voluntary motion in upper extremity following hemiple-

gia: Arch Phys Med Rehabil 46:567, 1965.

Beevor, CE: The Croonian Lectures. Adlard and Son, London, 1904.

Benton, LA, et al: Functional Electrical Stimulation: A Practical Clinical Guide. Rancho Los Amigos Rehabilitation Engineering Center, California, 1980.

Bierman, W and Licht, S (eds.): Physical Medicine in General Practice, ed. 3. Hoeber, New York, 1952.

Bobath, B: Adult Hemiplegia: Evaluation and Treatment. William Heinemann Medical Books, London, 1970.

Bobath, K and Bobath, B: Spastic paralysis treatment by the use of reflex inhibition. Br J Phys Med 13:121, 1950.

Brain, WR: Mind Perception and Science. Blackwell, Oxford, 1951.

Braun, et al: Surgical Treatment of the Painful Shoulder Contracture in the Stroke Patient. J Bone Joint Surg 53A (7):1307, 1971.

Brunnstrom, S: Movement Therapy in Hemiplegia: A Neurophysiological Approach. Harper & Row, New York, 1970.

Cailliet, R: The Shoulder in Hemiplegia. FA Davis, Philadelphia, 1980.

Carroll, D: Hand function in hemiplegia. J Chronic Dis 18:493, 1965.

Davies, PM: Steps to Follow: A Guide to the Treatment of Adult Hemiplegia, Springer-Verlag, New York, 1985.

Friedland, F: Physical therapy. In Licht, S (ed): Stroke and Its Rehabilitation. Williams & Wilkins, Baltimore, 1975.

Garrison, SJ: Update on heterotropic ossification in spinal cord injury. Current Concepts in Rehabilitative Medicine 5 (1):1, 1989.

Griffin, JW: Hemiplegic shoulder pain. Phys Ther 66 (12):1884, 1986.

Hakuno, A, et al: Arthrographic Findings in Hemiplegic Shoulders. Arch Phys Med 65:706, 1984.

Hettinga, DL: I. Normal joint structures and their reaction to injury. J Orthop Sports Phys Ther 1:1, 1979.

Hurd, MM, Farrell, KH, and Waylonis, GW: Shoulder sling for hemiplegia: Friend or foe? Arch Phys Med Rehabil 55:519, 1974.

Jackson, JH: On some implications of dissolution of the nervous system. Med Press Circ 2:130, 1951.

Jenson, EM: The hemiplegic shoulder. Scan J Rehabil Med (Suppl) 7:113, 1980.

Johnson, H and Garton, W: Muscle re-education in hemiplegia by use of electromyographic devices. Arch Phys Med Rehabil 54:1973, p. 320.

Johnstone, M: The Stroke Patient: Principles of Rehabilitation. Churchill Livingstone, London, 1976.

Johnstone, M: Restoration of Motor Function in the Stroke Patient. Churchill Livingstone, New York, 1978, p 15.

Kaplan, PE, et al: Stroke and brachial plexus injury: A difficult problem. Arch Phys Med Rehabil 58:418, 1977.

Knott, M: Introduction to and philosophy of neuromuscular facilitation. Physiotherapy 53:1, 1967.

Knott, M and Voss, D: Proprioceptive Neuromuscular Facilitation: Patterns and Techniques, ed 2. Harper & Row, New York, 1968.

Kottke, FJ: Neurophysiological Therapy for Stroke and Its Rehabilitation. In Licht, S (ed): Stroke and Its Rehabilitation. Waverly Press, Baltimore, 1975.

Licht, S (ed): Stroke and Its Rehabilitation. Waverly Press, Baltimore, 1975.

Najenson, T, Yacubovich, E, Pikielny, SS: Rotator cuff injury in the shoulder joints of hemiplegic patients. Scand J Rehabil Med 3:131, 1971.

Nicholas, J: Ectopic bone formation in patients with spinal cord injury. Arch Phys Med Rehabil 54:354, 1973.

Riedel, B: Demonstration eine durch achttagiges umhergehen total destruirten Kniegelnkes von einem patienten mit stichverletzung des ruckens. Verhanlüngen der Deutschen Gesellschaft für Chirurgie 12:93, 1883.

Risk, TE, et al: Arthographic Studies in Painful Hemiplegic Shoulders. Arch Phys Med 65:706, 1984.

Roper, BA: Rehabilitation after a stroke. J Bone Joint Surg 64B(2):156, 1982.

Ryerson, S and Levit, K: The shoulder in hemiplegia. In Donatelli, R (ed): Physical Therapy of the Shoulder. Churchill Livingstone, New York, 1986, page 105.

Schmidt, RF: Fundamentals of Neurophysiology. Springer-Verlag, New York, 1978.

Shah, SK and Corones, J: Volition following hemiplegia. Arch Phys Med Rehabil 61:523, 1980.

Tobis, JS: Post-hemiplegic shoulder pain. NY State J Med 57:1377, 1957.

Twitchell, TE: The restoration of motor function following hemiplegia in man. Brain 74:443, 1951.

Van Buskirk, C: Return of motor function in hemiplegia. Neurology 4:919, 1954.

Van Buskirk, C and Webster, D: Prognostic value of sensory deficit in rehabilitation of hemiplegics. Neurology 5:407, 1955.

Van Langenberghe, HVK, et al: Shoulder pain in hemiplegia: A literature review. Physiotherapy Practice 4:155, 1988.

VanOuwenaller, C, et al: Painful Shoulder in Hemiplegia. Arch Phys Med 67:23, 1986.

Voss, DE: Should patients with hemiplegia wear a sling? Phys Therapy 49:1030, 1969.

Waters, RL: Upper Extremity Surgery in Stroke Patients. Clin Orthop 131:30, 1978.

CHAPTER 10

Reflex Sympathetic Dystrophy

The condition termed *reflex sympathetic dystrophy* (RSD) now encompasses many neuromuscular vasomotor disabling conditions of both lower and upper extremities. This condition, aptly termed a *syndrome*, is known to follow virtually any form of local injury, major or minor. The condition was originally termed *causalgia* and implied *burning pain* with associated neurovascular symptoms. This syndrome is more significantly broken down to major and minor, in which pain—burning or otherwise—need not be associated. By so delineating the condition it is more apt to be recognized early and treated properly.

Bonica divided RSD into the categories major and minor in the following subdivisions:

REFLEX SYMPATHETIC DYSTROPHY

Major

Causalgia
Thalamic syndrome
Phantom limb

Minor

Shoulder-hand-finger syndrome
Post myocardial infarct
Post cerebrovascular attack
Postinjection
Post fracture, cast, splint,
Post overuse syndrome

It is apparent that in the above minor dystrophies any trauma can be implicated, which may indicate an incident so minor that the cause is difficult to remember.

That a major or minor RSD occurs in the upper extremity is the reason for indicating this syndrome in shoulder pain. Because RSD is a major complication in any upper extremity impairment, painful or painless, its recognition must be entertained in any complaint of the arm presented by the patient.

Perusal of the literature indicates that *burning pain* and associated symptoms following a peripheral nerve injury were noted by Paré in the 16th century (Bonica, 1990). It reached prominence during the American Civil War when it was described by Mitchell and associates. Mitchell described the condition in wounded soldiers who developed a burning pain following a peripheral nerve injury—usually from gunshot wounds. Mitchell employed the term *causalgia* for describing the burning character of the pain. Currently this condition is classified as major reflex sympathetic dystrophy.

Many clinicians followed with descriptive discussions of this condition. Letievant of France ascribed the condition to a neurologic origin. Sudeck published a classic description of the radiologic characteristics of bone osteoporosis following trauma with subsequent RSD. Leriche described the condition as a sequela of the sympathetic nervous system which led to peripheral sympathectomy as a favored and successful treatment of RSD. Reflex sympathetic dystrophy was largely forgotten until World War II when numerous cases were reported and the clinical condition revived.

Numerous terms have been used in the literature, such as algodystrophy, sympathalgia, neurovascular reflex sympathetic dystrophy, traumatic angiospasm, traumatic vasospasm, Sudeck atrophy, posttraumatic osteoporosis, posttraumatic painful osteoporosis, and shoulder-hand-finger syndrome. Often pain persists after the initial trauma subsides; it remains in the peripheral nerve transmitted by sympathetic nerve fibers. This classification of persisting pain has been labelled *sympathetically maintained pain* (SMP). If there is less evidence of sympathetic nervous system involvement—yet persistence of the pain—the term *sympathetic independent pain* (SIP) is invoked (Roberts).

All the above refer to clinical manifestations:

1. Pain: variously described
2. Vasomotor changes: subjective and objective
3. Functional impairment: muscular, dermal, and articular
4. Ultimate osteoporosis
5. Atrophic osteoarthrosis

Causalgia has been defined by the International Association for the Study of Pain (IASP) as "a syndrome of sustained burning pain after traumatic nerve lesion combined with vasomotor and sudomotor dysfunction and later trophic changes." Amplification of the RSD syndrome (Bonica, 1979a) now lists many conditions of RSD without *burning pain* but with all the vasomotor and sudomotor symptoms and findings. This would conform to SIP pain advocated by Roberts. The basic mechanisms, pathophysics, and symptoms (Tahmouth) are similar enough in any form of RSD to justify the diagnostic term and therapies.

A definition of the terms of RSD clearly clarifies this disease entity. *Dystrophy* indicates wasting of the muscular and bony tissues of the region as well as abnormal growth of the nails of the extremities and hyperkeratosis of the skin. *Sympathetic* indicates vasomotor and sudomotor changes, such as inappropriate sweating, coldness, and color changes of the extremity from vasoconstriction or vasodilatation. *Reflex* signifies that the signs emanate from the sympathetic nervous system distribution of the extremity. Another confirmatory diagnostic fact of RSD is its beneficial response to sympathetic interruption.

Onset of pain varies between major RSD (causalgia) and minor RSD in that the former has an immediate onset of pain or at least one within a brief period of time, whereas minor RSD may have a delay of pain of several days to months. The character of pain must be of a burning quality to qualify as being RSD.

The site of this syndrome is distal in the extremity: whether it is upper shoulder-arm-hand-finger or lower knee-ankle-foot-toes.

Postulated mechanisms have varied. Mitchell (1872) proposed "an inexplicable reflex in the spinal cord centers felt in remote regions outside the distribution of the wounded nerve." I have modified this definition. Numerous concepts have been postulated, but a more recent one offered by Devor suggests that the injury damages or transects (cuts across) the involved nerve.

This appealing theory involves understanding the structure of nerve axons

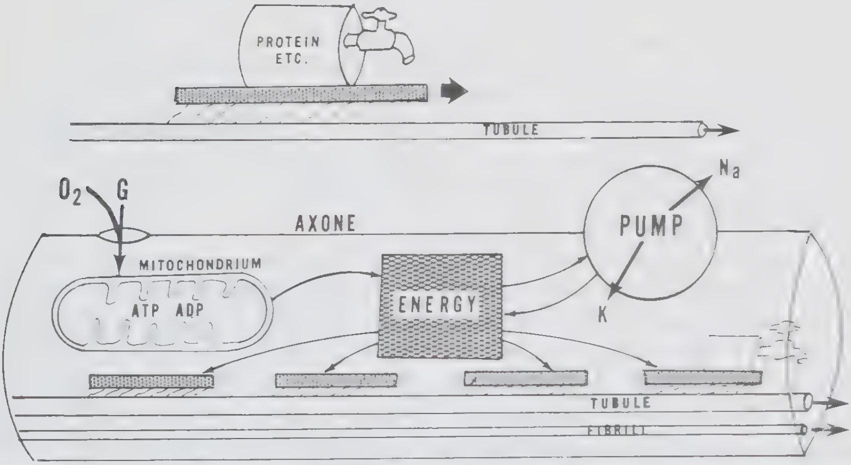


Figure 10–1. Axoplasmic neural transport: a theory. The *flow* of protein and other derivatives begins with entry of glucose (G) into the fiber. Glycolysis and phosphorylation occur (O_2) in the mitochondria through metabolism of adenosine-triphosphate (ATP), which creates the energy to the sodium pump. This pump regulates balance of sodium (N_a) and potassium (K) and determines nerve activity.

The transport *filaments* (F) move along the axon by occillation and carry the nutritive protein elements along the nerve pathway. (Data from Ochs, S: Axoplasmic transport: A basis for neural pathology.)

and the theory of axoplasmic transport. The hypothesis is exemplified in Figure 10–1.

Neuronal function is now considered to be axonal transport of protein and other materials needed by the tissues supplied by the nerve. Sensory impulses are also supplied by axonal transport.

The neuron cell body undergoes a high level of protein synthesis, which is conveyed along the length of the nerve fiber. This transport mechanism has been shown to be very dependent on adequate blood supply. Pressure on the nerve axon and/or its blood flow will impair axonal transport. The flow through the axon microtubules and neurofilaments is impaired. Variation of the components of the proteins of the peripheral nerve will also determine the end result of axonal impairment.

After a nerve is constricted, the fibers may show collateral branching (Perroncito, Shawe). During recovery of the injured nerve, the exposed regenerating surface of the axon undergoes more than normal accumulation of receptors (Devor). These receptors are alpha-adrenogenic, resulting in abnormal electrical properties of that nerve (Wall and Gutnick). These excessive, and possibly abnormal, receptors become ectopic pacemakers that lead to spontaneous depolarization. Numerous and excessive, they bombard the central nervous system and interfere with normal central processing of sensory information. The central nervous system, already in a state of hyperactivity and hyperreceptivity from previous bombardment by unmyelinated nerve fiber impulses, is now more accessible to persistence of pain by this added excessive release of distal adrenaline impulses from these new branchings of the nerve fibers (Devor) (Fig. 10–2).

The peripheral stimuli originate because of abnormal chemosensitivity and mechanosensitivity of the neurons and not as a result of other physiologic stimuli.

Centrally the aberrant sensory processing from this barrage produces a sensation of pain (paresthesia), and the altered sympathetic reflexes produce the somatic characteristics of RSD (Fig. 10–3).

There are theories involving the peripheral nervous system, other than those regarding central mechanisms. At the site of the nerve injury there is a synapse between efferent sympathetic and afferent pain fibers that *short circuits* the sensory information (Doupe and colleagues). This concept has been refuted because it does not explain why a sympathetic nerve block distal to the lesion is effective. Another peripheral concept invokes liberation, at the involved area (Leriche), of algesic substances (nociceptive) that produce local hyperalgesia, which, in turn, produces and sustains a vicious cycle.

Of the numerous *central* mechanisms postulated, the concept of bombardment of the dorsal horn cells in laminae IV to VI by the peripheral nociceptor substances is widely held (Fig. 10–4).

As stated by Rizzi and associates, an unanswered question is posed in why causalgia occurs most often with a *partial* nerve lesion rather than with a complete lesion and why there is such a low incidence of causalgia with so many partial nerve lesions.

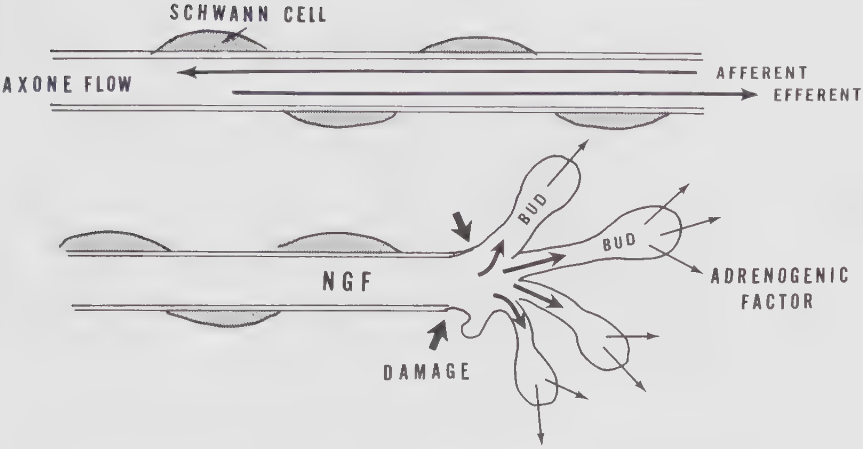


Figure 10–2. Axonal outgrowths forming a neuroma (schematic). After a nerve injury with compression or partial to total severance, the nerve growth factor (NGF) stimulates the nerve to advance distally and form "buds", which create more endings than the normal nerve shown in the upper drawing.

By virtue of the greater secretion of adrenogenic factors, the nerve becomes more sensitive to adrenogenic agonists and transmits more potential pain fiber impulses to the spinal cord (see also Fig. 10–5).

The psychologic state of the individual at the time of injury has also been proposed as a significant factor (Wirth and Rutherford; Owens). Research still is needed to ascertain the neuro-psychologic-humeral susceptability of individuals under extreme anxiety that predisposes patients having trauma to develop causalgic RSD.

A hypothesis has recently been advanced (Ecker) for the fact that many cases of RSD occur after a relatively minor trauma and that pain and other somatic symptoms are exacerbated by emotional stress. He postulates that preexisting anxiety and/or stress increases the release of norepinephrine, which increases arteriolar hyperactivity. The resultant vasospasm, ischemia, and nociceptor release upon neural tissues already bathed by excessive norepinephrine from the anxiety and/or stress results in RSD.

There is also aggravation of the local pain and referred pain often from minor, unrelated, and reasonably innocuous stimuli, which are transmitted to the central nervous system via mechanoreceptors. These receptors and their afferent fibers normally do not transmit pain, but in this condition, SMP, they enhance the pain. This concept is illustrated in Fig. 10–5.

The original trauma transmits action potentials through C-nociceptive fibers to the dorsal root ganglion (DRG), where they are transmitted to the cord in the region of the dorsal horn (rexed layers). They bombard that region and form a hyersensitive set of neurones (termed WDR by Roberts). This WDR can

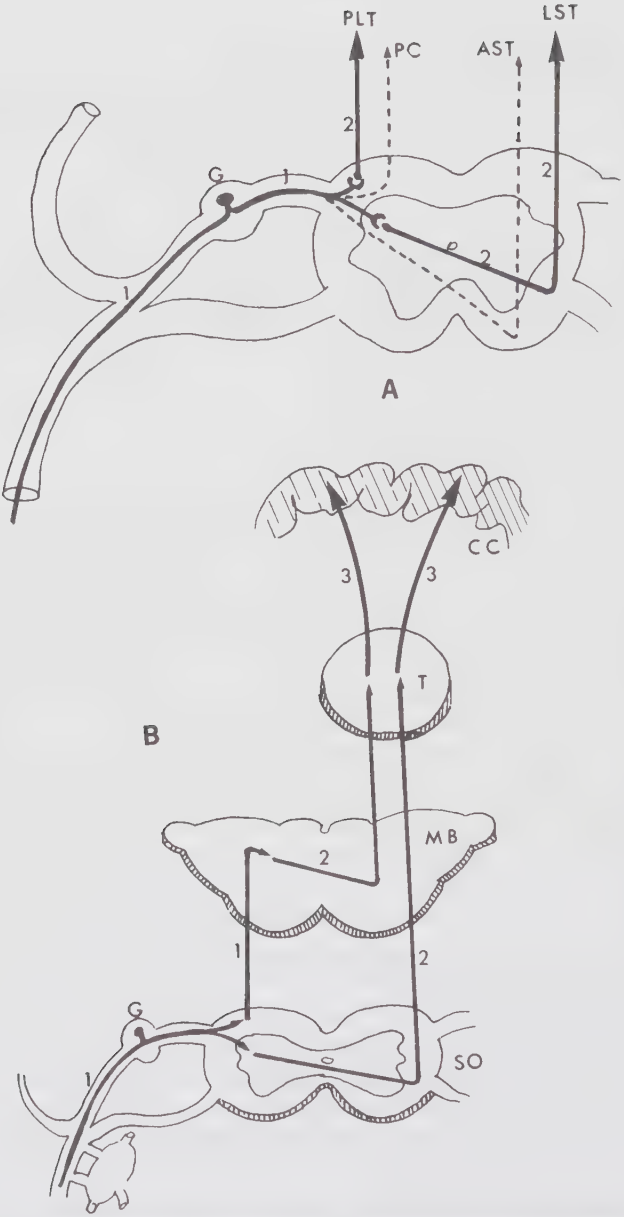


Figure 10–3. Neuron pathways of pain. *(A)* The course of sensory fibers in a segmental nerve with its ganglion in the dorsal root (G). Upon entrance into the cord, the fibers ascend on the same side in the posterior lateral tract (PLT) and decussate to cross into the lateral spinothalamic tract; 2 indicates secondary neurons. The posterior column (PC) transmits position sense; AST conveys tactile sensation. *(B)* 1 = first-stage neurons to the cord; 2 = second-stage neurons through the midbrain (MB) into the thalamus (T); 3 = third-stage neurons, the thalamocortical pathways to the cerebral cortex (CC).

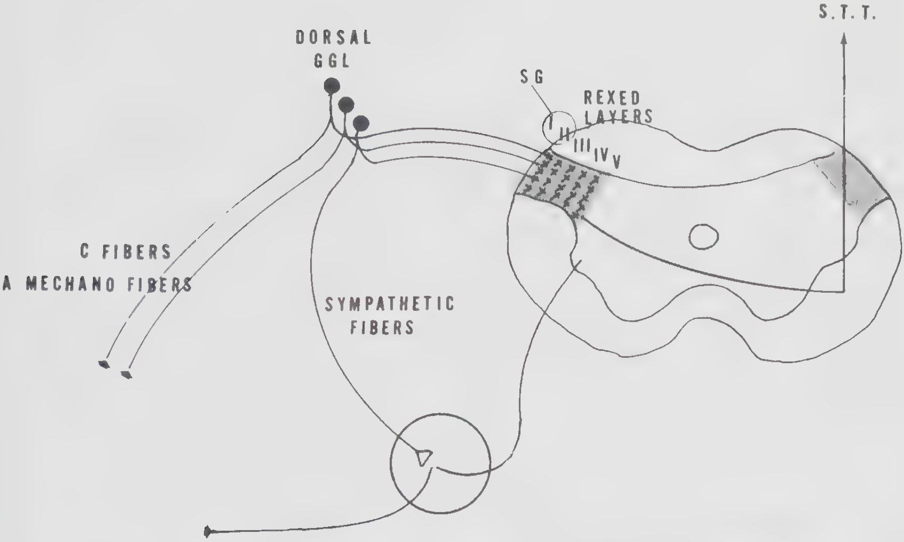


Figure 10–4. Causalgic (autonomic) transmission of pain sensation. Trauma irritates somatic afferent C fibers, A mechanofibers, and sympathetic fibers, whose impulses proceed to the dorsal column of the cord. There is a cord interneuron connection that transmits efferent impulses via the autonomic system to the periphery, which sensitizes the skin to mechanical (light touch) input.

At the cord, the afferent fibers initiate neuronal activity in the Rexed layers of the dorsal column. The Rexed layers I and II are the substantia gelatinosa. (Modified from Roberts, WS: A hypothesis on the physiological basis for causalgia and related pains. Pain 24:297, 1986. From Cailliet, R: Soft Tissue Pain, ed 2. FA Davis, Philadelphia, 1988, with permission.)

then be bombarded by impulses via the mechanoreceptors from the skin, muscles, tendons, and ligaments that travel via A-mechano fibers of myelinated nerves. These later impulses impinge upon the already hypersensitive cord regions (WDR) that can "spill" over, or influence the proximal nerve cells (lateral horn) of the autonomic (sympathetic) nerves. This indicates how inocuous unrelated touch pressure or movement can intensify the pain even though these are mechanoreceptors and not nociceptors. These impulses travel distally (efferent) to the periphery causing vasomotor reactions as well as stimulating sympathetic pain sensations.

The vasomotor changes of the dystrophy that evolve are thus explained. Also explained is the basis for sympathetic afferent impulses initiating the cycle, which is enhanced by mechanoreceptor irritants (i.e., touch, stretch, passive and active motion).

In the symptomatic shoulder lesions that eventually develop a minor or major RSD, the etiologic concept may remain unanswered, but its occurrence de-

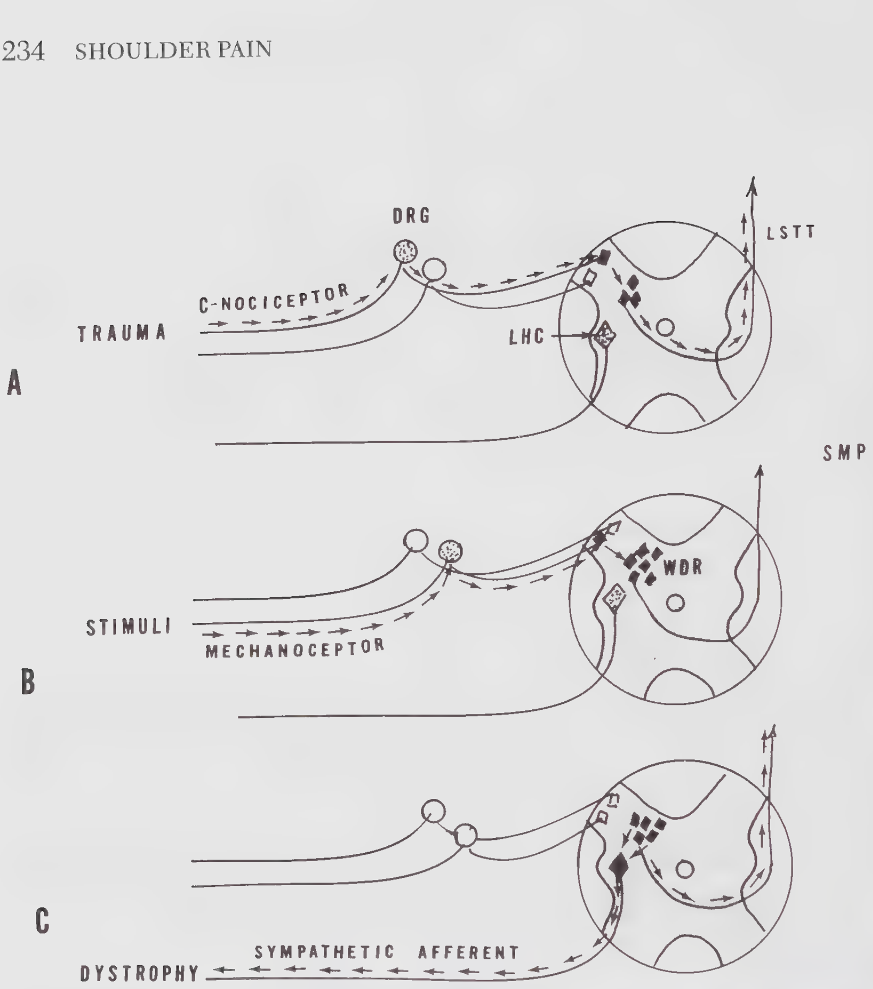


Figure 10–5. Postulated neurophysiologic mechanism of sympathetic maintained pain (SMP). The transmission via C-nociceptor fibers (*A*) of impulses from the peripheral tissues that have been traumatized and created peripheral nociceptor chemicals (see details in text). These impulses pass through the dorsal root ganglion (DRG) to activate the grey matter of the cord in the rexed layers. When sensitized, they are termed *wide dynamic range neurones* (WDR). The WDR, becoming very irritated, receives impulses from the periphery via the A-mechanoreceptors fibers, (*B*) which normally transmit sensations of touch, vibration, temperature, etc. When the periphery is stimulated, (skin touch, pressure, or joint movement) these impulses enhance and maintain the irritability of the WDR. The impulses from the WDR continue cephalad through the lateral spinal thalamic tracts (LSTT) to the thalamic centers with resultant continued pain. The WDR impulses irritate the lateral horn cells (LHC), which generate sympathetic impulses that innervate the peripheral tissues resulting in the symptoms and findings of dystrophy (*C*).

mands attention—diagnostic and therapeutic—to minimize the disabling sequelae.

REFLEX SYMPATHETIC DYSTROPHY (RSD) IN SHOULDER LESIONS

Whereas major RSD causalgia occurs posttraumatically in shoulder injuries, by definition there must be a partial nerve lesion for RSD and causalgia to occur. In conditions such as shoulder dislocations, adhesive capsulitis, and poststroke hemiparetic shoulder lesions, no nerve lesion is needed to have RSD develop. The minor RSD lesion of shoulder-hand-finger syndrome, regardless of the origin, usually does not have a partial nerve lesion or develop a painful causalgia. But all the sequelae of RSD do develop.

SHOULDER-HAND-FINGER SYNDROME

In this condition there is dysfunction of the sympathetic nervous system but on a different physiologic basis and from a different origin. As a rule, this RSD is mechanical, from a vascular basis.

The normal circulation of the upper extremity can be simplistically divided into arterial and venous components, both of which have a mechanical component.

Arterial Component

The arterial component is composed of the cardiac *pumping* action, major arterial tone, and constriction-relaxation cycle, and the gravitational forces that propel the arterial blood flow to the distal portions of the upper extremity. The blood flow through the major arteries and then arterioles ends in the capillaries, where there is diffusion into the tissues.

Venous Component

The return of the circulation to the heart and lungs is by way of the venous and lymphatic channels by virtue of *pump* action. The muscles of the forearm, hand, and fingers literally pump the blood proximally with the assistance of gravity.

The arm must be held frequently above heart level for gravity to be effective. The shoulder girdle muscles thus act as a proximal pump in this activity. The shoulder girdle muscles move the arm in every direction and, in addition to pumping the venous lymphatic and blood elements toward the heart, elevate the upper extremity above heart level (Fig. 10–6).

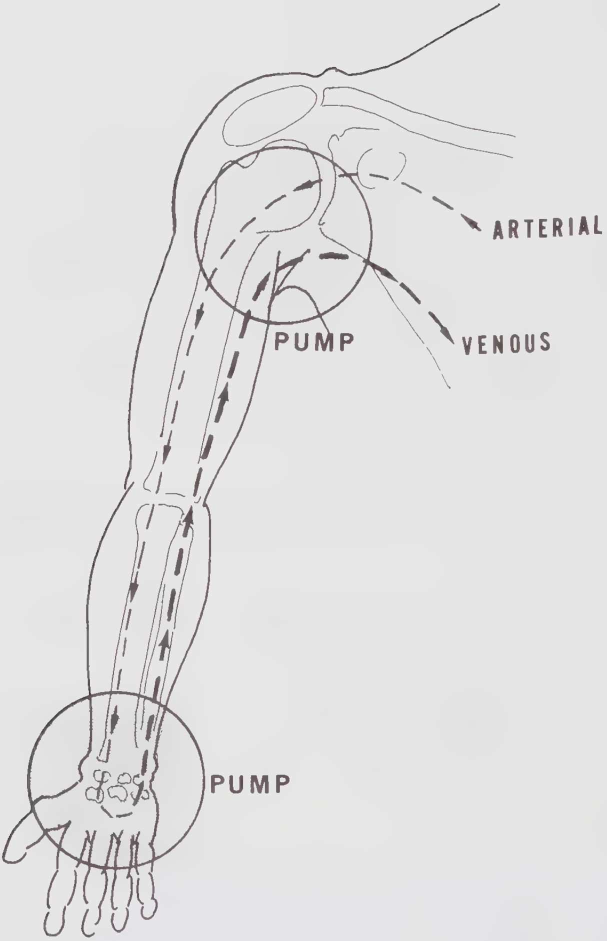


Figure 10–6. Venous lymphatic pumps of the upper extremity.

Repeated clenching and relaxing muscular action of the hand and fingers pump the blood and lymphatic fluid proximally. The largest portion of the arterial supply of the hand and fingers is in the volar aspect of the hand, and the venous-lymphatic drainage is in the dorsal tissues of the hand and fingers.

Failure of either of these pumps, shoulder or hand, to function adequately may lead to a painful and disabling condition termed *shoulder-hand-finger syndrome.* Loss of hand-finger alternating flexion-extension eliminates the distal pump. It is well known that a fracture-dislocation of the wrist or fingers with or without casting may initiate this condition.

Pain from this syndrome varies from an *ache,* deep discomfort, tenderness, painful movement, or even mild *burning*. Pain usually does not occur initially nor necessarily early in this entity but may be noted several days to weeks after the onset. Most often there are impairments of function other than pain, which is why the condition is not diagnosed early in many cases.

Mechanism of Shoulder-Hand-Finger Syndrome

The sequelae of the shoulder-hand-finger syndrome as related to the shoulder is that, as a result of whatever initial condition, the shoulder pump does not function. The arm fails to move appropriately, and ultimately there is failure of elevation above the heart level. Inadequate shoulder girdle motion impairs the upper pump action, and there is diminution of venous lymphatic flow.

There are many factors that may initiate limited shoulder action, among which are

1. Adhesive capsulitis
2. Fracture-dislocation of the glenohumeral joint
3. Painful supraspinatus tendinitis
4. Posthemiplegic shoulder impairment

The stages of shoulder-hand-finger syndrome are depicted in Figure 10–7.

Diagnosis of Shoulder-Hand-Finger Syndrome

The complex of shoulder-hand syndrome described by Osler in 1897 following a myocardial infarction was formulated in 1948 by Steinbrocker and associates. Subsequently, regardless of the site and source of the initial pain in the shoulder region, the sequelae were noted to be identical.

The syndrome may originate in both shoulders but usually occurs in one. Except in the case of myocardial infarct, it occurs more often in women. The shoulder is usually involved first, with the hand and fingers following, but, as in the case of case of a Colles wrist fracture, the opposite sequence can occur.

The shoulder may become *stiff* in that there is limited passive and active range of motion and its cause must be addressed. Most of these causes have been discussed in the text and they are as follows:

1. Supraspinatus tendinitis
2. Rotator cuff tear
    a. Partial
    b. Complete
3. Adhesive capsulitis

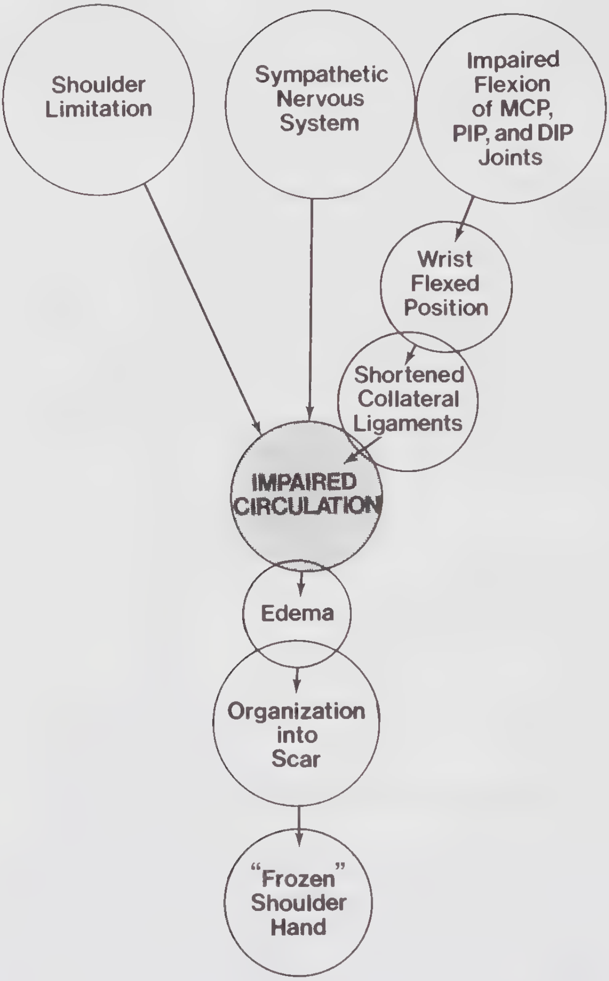

Shoulder Limitation
Sympathetic Nervous System
Impaired Flexion of MCP, PIP, and DIP Joints
Wrist Flexed Position
Shortened Collateral Ligaments
IMPAIRED CIRCULATION
Edema
Organization into Scar
"Frozen" Shoulder Hand

Figure 10–7. Sequence leading to *frozen* shoulder-hand-finger syndrome.

4. Postcerebrovascular hemiplegia
5. Post fracture-dislocation within the scapular humeral complex
6. Shoulder paresis from a peripheral nerve injury
7. Systemic paresis, such as Guillain-Barré syndrome, poliomyelitis
8. Spinal cord injury with paraplegia or quadriplegia
9. Inappropriate immobilization from casting or sustained position

The onset of the RSD shoulder-hand-finger syndrome is first noted in the hand, where there is subtle edema. The initial evidence of this edema is noted on the dorsum of the fingers, where there is the preponderance of venous and lymphatic vessels. The skin becomes shiny, smooth, and pale. The wrinkles of the knuckles and each of the joints are erased. Pitting may be elicited, but the degree of pitting is usually so minimal that it may escape attention.

Full flexion is decreased. Evaluation requires careful attention to the degree of limitation, so minimal that it must be compared with that of the opposite hand or its presence will escape attention. This limited range of motion is the subtle beginning of the loss of the distal pump.

The diminution of finger flexion is caused by the edema under the extensor

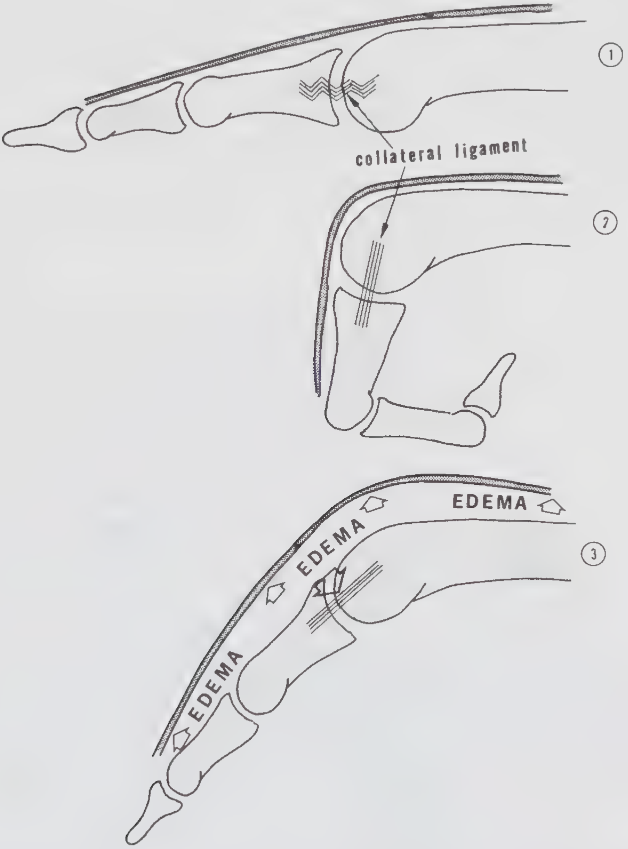


Figure 10–8. Finger changes in the hand-shoulder syndrome. *(1)* Normal extension of the metacarpophalangeal joint with relaxed collateral ligament. *(2)* Normal flexion of the metacarpophalangeal joint with the collaterals becoming taut. *(3)* Edema on the dorsum of the hand elevates the extensor tendons and prevents flexion. The collateral ligaments are never fully elongated and develop contracture. This further limits the *pump action* of the flexion of the hand.

tendons (Fig. 10–8). The elevation of these tendons decreases their mechanical efficiency.

Ultimately the edema spreads to (under) the collateral ligaments, which further impairs full flexion of the fingers. Normally the collateral ligaments are slack when the fingers are in full extension. This allows abduction-adduction of the digits. Because the distal ends of the metacarpals and the phalanges are ovoid (forming an incongruous joint), the collateral ligaments become taut as the distal fingers flex (Fig. 10–9).

If the collateral ligaments become taut by virtue of edema forming under them with the fingers extended, no flexion is permitted. When the collateral ligaments are made taut with the fingers extended, they have insufficient laxity to permit the proximal aspect of the next distal phalanx to glide, in flexion, upon the ovoid distal joint surface of the proximal joint surface. The distal hand pump is further diminished and ultimately eliminated.

At first edematous and ischemic, the skin becomes thickened, then ultimately atrophic. Clinically there is early hyperhydrosis (excessive sudomotor activity). The hand is moist. It may be either pale or with slight rubor. The color

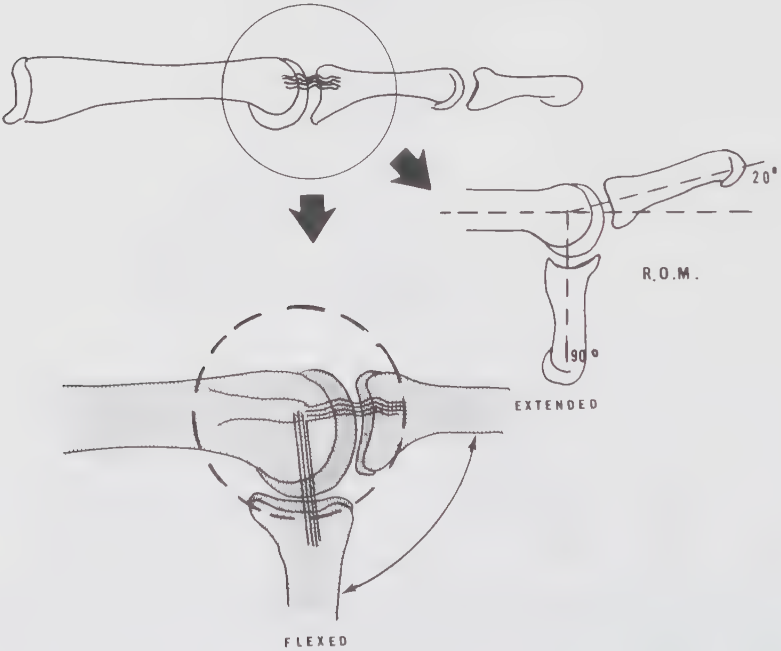


Figure 10–9. Normal flexion-extension of the metacarpophalangeal joints. Due to the elliptical shape of the head of the metacarpals, the collateral ligaments are slack with finger extension and taut when the fingers are flexed.

changes depend on impaired vasomotor tone as to whether there is vasodilatation or vasoconstriction. The hand is thus moist, pale, cold or warm, and, when compared with the normal hand, the vasomotor-sudomotor abnormality is noted early in the condition.

Although the affected skin in RSD is more often cold (vasoconstricted) than warm (vasodilatation), there is an *increased* blood flow in the subcutaneous tissues (Christensen and Henricksen), muscles (Sylvest and colleagues), and bones (Ficat and associates). This increased bone blood flow may account for the increased activity noted in radioactive bone scans in RSD. This increased deeper blood flow also may initiate a transient arteriovenous shunt with decreased superficial blood flow, hence the ultimate dystrophy.

The hair follicles thicken (hypertrichosis), as well as the nails, from excessive sudomotor activity. All sudomotor and vasomotor changes are subtle at first and progress gradually. The stage at which they are discovered and the point at which treatment is started determine the correctability or reversibility of the structural changes.

Stages of Shoulder-Hand-Finger Syndrome. Stage 1: Vasomotor signs of hyperhydrosis with edema

- Limited shoulder range of motion (with or without pain)
- Swelling of dorsum of hand at first pitting
- Skin becomes shiny, dry, or moist
- Limited range of motion of finger flexion
- Pain on wrist extension

Stage 2: Most significant change in the edema is that it is *firmer* and cannot be dimpled by pressure.

- Shoulder pain may subside, and there may be slight increase of active and passive range of motion.
- Edema of hand appears to subside but is less pitting
- Skin is less elastic; there are no wrinkles at the metacarpal-phalangeal or distal phalangeal joints; the hand is usually now cold and dry
- Fingers become stiffer
- The nail and hairs become coarser
- Skin becomes less sensitive
- Osteoporosis becomes evident on x-ray film

Stage 3:

- There is progressive atrophy of bones, skin, and muscles
- There is limited passive range of motion at the metacarpal-phalangeal and distal phalangeal joints
- The nails are brittle and grooved; the hair follicles are large and brittle

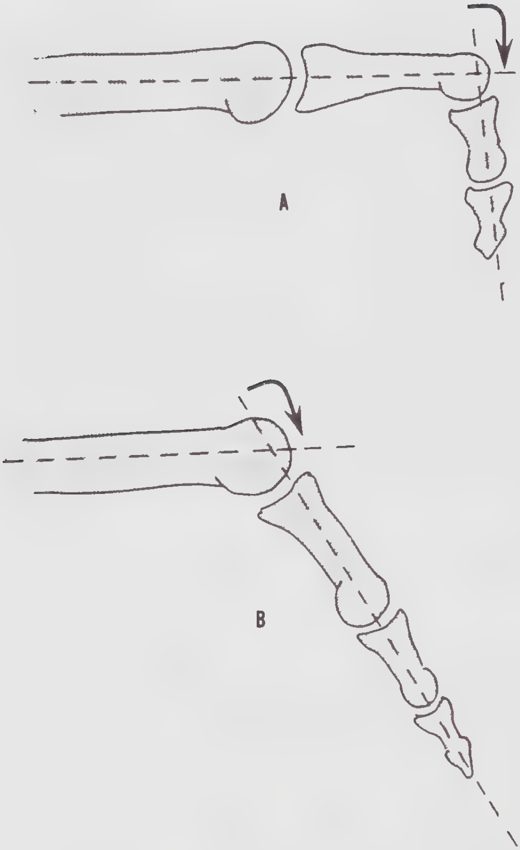


Figure 10–10. Hand patterns. *A* depicts the hand that resembles the "intrinsic minus hand." The proximal phalanx remains extended with tenodesis action flexing the distal phalanges. *B* resembles the "intrinsic minus hand," with all phalanges extended but with the metacarpophalangeal joint flexed.

- Pain may now be minimal or absent except when passive motion is attempted
- The hand may resemble the *intrinsic minus hand* (Fig. 10–10)

X-ray studies that reveal bone atrophy (osteoporosis) may be noted early even at stage 1, but usually it is noted at stage 2. Diagnostic x-ray studies should always include the opposite hand for comparison because early changes are subtle. Bone density studies have currently been developed that differentiate and grade the degree of osteoporosis, but this issue is more academic inasmuch as it must be stated that the initial diagnosis *must not be made upon finding bone density changes in hand x-ray studies.* By then stage 3 is in its early onset.

During the progression of shoulder-hand-finger syndrome there are ultimately atrophic articular changes. The cartilage of the hand joints—carpal, metacarpal, and phalangeal—impairs the circulation of the joints by virtue of the ischemia from vasomotor abnormality, and atrophic arthritis results. The hand and fingers now are held in a *clawlike* status and no passive or active motion is possible. There is no pain, but there is also no function of the hand and wrist.

Stages 1 and 2 are considered reversible to a practical functional degree. Stage 3 has many irreversible structural changes that make functional recovery limited, if at all feasible.

Diagnosis should be suspected when there are subtle skin changes of the hand in any condition of the upper extremity in which there is

1. A shoulder *problem* of pain and limitation
2. Pain or limitation of the elbow and wrist
3. Pain and limitation of the digits of the hand
4. Trauma to the upper extremity such as surgery, injection, or a sprain-strain
5. A systemic condition with referred pain to the upper extremity.

Treatment

Treatment of RSD varies only slightly when there is causalgic pain or not. Causalgic pain must obviously be addressed forcefully and energetically until overcome or significantly minimized. Without relief of causalgic pain the syndrome cannot be remedied or moderated. Treatment of the sequelae of reflex dystrophy must also be simultaneously and energetically addressed concurrently with treatment of causalgic pain. To relieve the pain and have a residual stage 3 hand would ill serve the patient.

Interruption of sympathetic hyperactivity is universally indicated. For RSD of the upper extremity, a chemical block of the sympathetic nervous system at the stellate ganglion was originally considered diagnostic and therapeutic. Other forms of sympathetic intervention have subsequently been advised, but the chemical stellate block still prevails as the initial treatment.

White and Sweet suggested that, because of the prevalence of emotional factors in this condition, a placebo diagnostic test should be considered. Upon getting relief after a local anesthetic but no response from sterile saline, sympathetic interruption should be considered. Because the condition is so ominous and stellate block (Fig. 10–11) is relatively simple and safe, initial active treatment should be considered and undertaken. If severe emotional problems play a part, therapy in that direction becomes a part of the treatment regimen.

As series of stellate blocks should be considered—usually a minimum of four, but more have been undertaken beneficially before a surgical extirpation of the stellate ganglion is considered. The decision is based on subjective and objective results of the chemical sympathectomy and the duration of the bene-

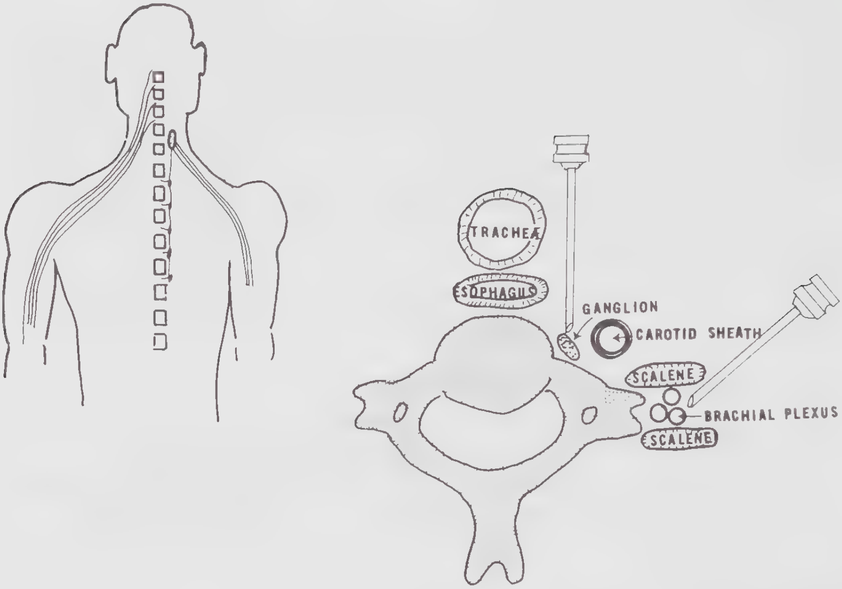


Figure 10–11. Technique of stellate ganglion or brachial plexus block. For a stellate ganglion nerve block, the trachea is moved to one side and the needle enters between it and the carotid artery to the vertebral body. The needle then is moved slightly lateral to the body, and 3 to 5 ml of the anesthetic agent is administered. Aspiration must always precede infiltration of the anesthetic agent.

fit. This is a clinical judgment based on the experience of the treatment physician.

A stellate block is considered effective if there is

1. Relief or diminution of pain
2. A Horner's syndrome, which indicates that the sympathetic chain has been interrupted (these signs are miosis, enophthalmos, and ptosis). Anhidrosis, injected conjunctiva, slight flushing of the face, and blocked nostrils are also frequently noted
3. A marked rise in the skin temperature of the extremity

Most benefits of the initial stellate block are lost or diminished within 24 to 48 hours and another block needs to be performed. Usually the next blocks are considered at 5- to 7-day intervals.

More permanent benefit from sympathetic intervention can be anticipated as follows:

1. Good
    a. If one block gives total relief
    b. If one block gives significant relief of pain
    c. If after a beneficial first block subsequent blocks give further significant relief.
2. Poor
    a. If the first block has beneficial effect but each subsequent block is less effective or totally ineffective
    b. If the initial benefit of the block wears off after the anesthetic effect is gone. This fact questions, in part, the value of adding a steroid agent to the anesthetic. The final answer to this has yet to be confirmed. A longer-acting anesthetic agent is probably of greater value than the addition of steroids.

If there is benefit from chemical interruption of the sympathetic agent but no continued benefit, a surgical sympathetic interruption should be considered. Here, however, the residual effects such as persistent ptosis, enophthalmos, miosis, and even some cosmetic facial *drooping* must be understood and accepted by the patient.

Oral or intramuscular steroids have been advocated and found beneficial. As in prescribing any medication, the undesirable side effects of any drug should be weighed against the hoped-for value of the medication. Large doses of a steroid may be valuable for a brief duration and need to be monitored carefully, weighing the benefit against the undesirable side effects.

Patients with severe intractable causalgic type of pain who do not or cannot beneficially avail themselves of stellate blocks may benefit from intravenous guanethidine (Hannington-Kiff). This drug is administered intravenously into the afflicted extremity with a cuff applied to restrict the effect of the guanethidine to that portion of the extremity. Intravenous reserpine, which is a vasodilator, has also been advocated (Benzon and others). All these intravenous treatments should be administered in a hospital setting to avoid adverse immediate or latent reactions.

The use of TENS has been found effective, as has acupuncture. Both are worth a trial period, so long as they are not used in isolation or after a reasonable time trial has proven of limited benefit.

In addition to benefit from stellate blocks and/or steroid medication, every local factor of the syndrome must be energetically attacked. The edema must be eliminated before it becomes indurated.

This requires elevation of the arm and hand above heart level as often as every half hour during the day. With a limited range of shoulder motion, from pain or from adhesions, this presents a problem.

Every modality of regaining shoulder range of motion, which has been discussed in previous sections, must be employed. This includes

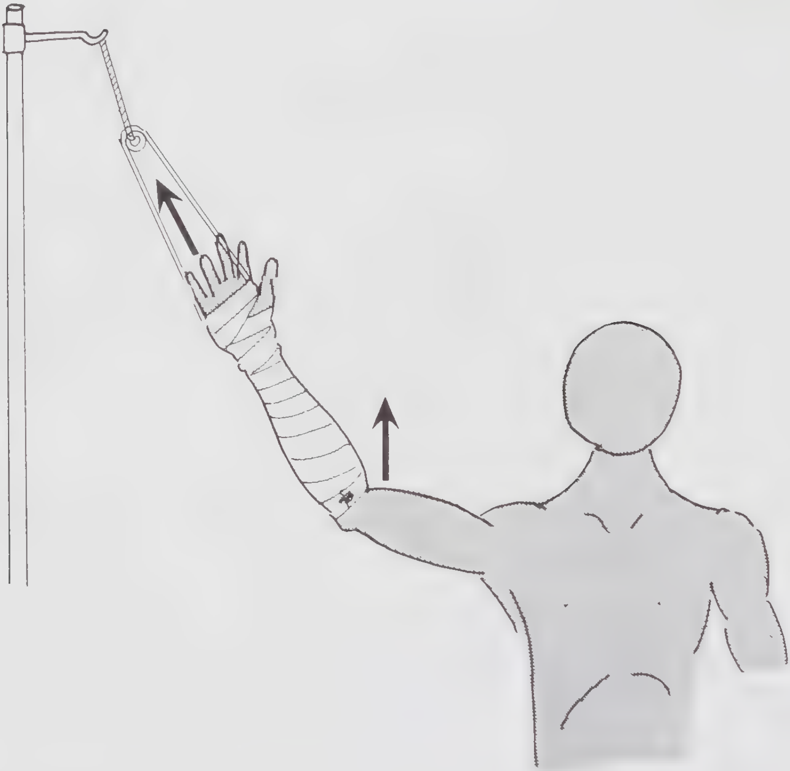


Figure 10–12. Antigravity treatment of the edematous extremity. The hand and arm are wrapped, distal to proximal, with an elastic bandage. The hand held by the webril is then suspended overhead. This position drains the edema and maintains shoulder range of motion with the elbow extended.

1. Passive range of motion exercises
2. Active range of motion exercises
3. Electrical stimulation
4. TENS unit application
5. Ultrasound to the shoulder
6. Activity of daily living position, such as a lapboard in a stroke patient or an overhead mechanical appliance (Fig. 10–12)

Removal of hand edema is also mandatory. This can be done in many ways, for example, retrograde wrapping (Fig. 10–13).

Manual *milking* of each finger and the total hand by a therapist is also effective. This can be done by the patient using the contralateral normal hand or, in a disabled patient, instructing a member of the family. The use of Jobst pneumatic equipment is also of value.

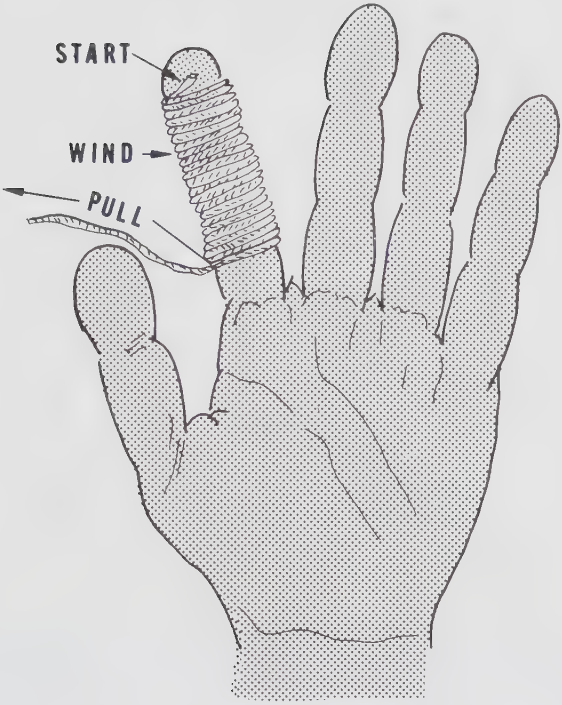


Figure 10–13. Removal of finger edema. Each finger is firmly wrapped with a heavy twine, beginning at the tip and moving toward the webbing. This procedure should be performed several times daily and can frequently be done by the patient, using the uninvolved upper extremity.

In a stroke patient in either the flaccid or the spastic stage (see Chapter 9 on the Hemiplegic Shoulder), proper and frequent positioning of the hand, wrist, and fingers is of value. The attempt in these positions is to ensure wrist and finger elevation. To mobilize merely the fingers and not the wrist will fail to gain full recovery.

Because there is usually a significant vasomotor component in the shoulder-hand-finger syndrome, contrast baths are valuable. These may be used individually or in addition (preceding and following) to active passive exercises. Two plastic buckets are used. One is filled with shaved ice, and the other, with warm water. Ice can be tolerated only for brief submergence but however long the patient can tolerate it will be beneficial. Active motion within the ice water is recommended. Immediate transfer to the warm water then follows, where the hand is actively and passively mobilized within the water. Obviously the warmth of the water must be tested by the normal hand before submergence of the affected hand.

Because ultimate atrophic ischemic arthritis changes can occur in the joints of the wrist and fingers, full range of motion must be undertaken. Each joint must

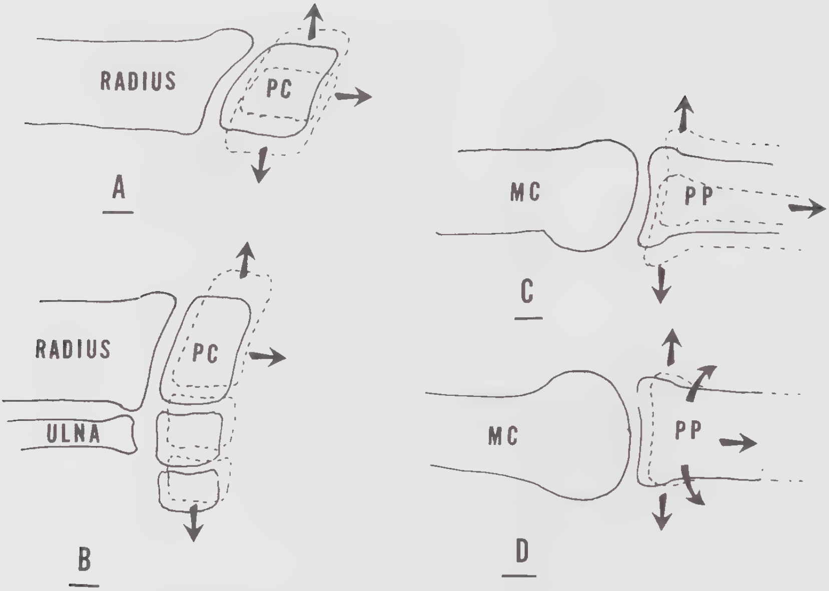


Figure 10–14. Mobilization of the joints of the wrist and fingers. Mobilization of the joints of the wrist and fingers utilizes the concept of joint play. The horizontal arrows depict the traction upon the joint, and the vertical arrows, the translation joint mobilization. The curved arrows in *D* depict rotation mobilization along with translation and traction.

A is a lateral view of the wrist with PC representing the proximal carpal bones. *B* is a vertical view of the wrist, revealing the radius and ulna articulating with the proximal carpal bones. *C* is a lateral view of the metacarpal (MC) joint with the proximal phalanx (PP). *D* is a vertical view of the metacarpal-proximal phalangeal joint depicting traction, translation, and rotation about the axis of the joint.

Manual mobilization requires the wrist to be held firmly while the mobilizing hand holds the distal bones of the joints being mobilized.

be stretched manually; the wrist in all directions, the metacarpal joints, and each of the interphalangeal joints must be mobilized. This mobilization must include traction, translation, and lateral motion as well as flexion-extension (Fig. 10–14).

Because the shoulder is usually limited in range of motion and partially or completely frozen, mobilization of the shoulder (Fig. 10–15) may also be needed as well as mobilization of the wrist and finger joints.

Active exercises are ultimately the most valuable modality of any therapy to regain function and to decrease pain. Use of plastic putty, warm sand, warm water, or rubber bands to give warmth and resistance to all finger motion is valuable home therapy. An occupational therapist is extremely valuable in outlining, directing, and supervising a good clinic and home program.

The consequences of shoulder-hand-finger syndrome also involve atrophy

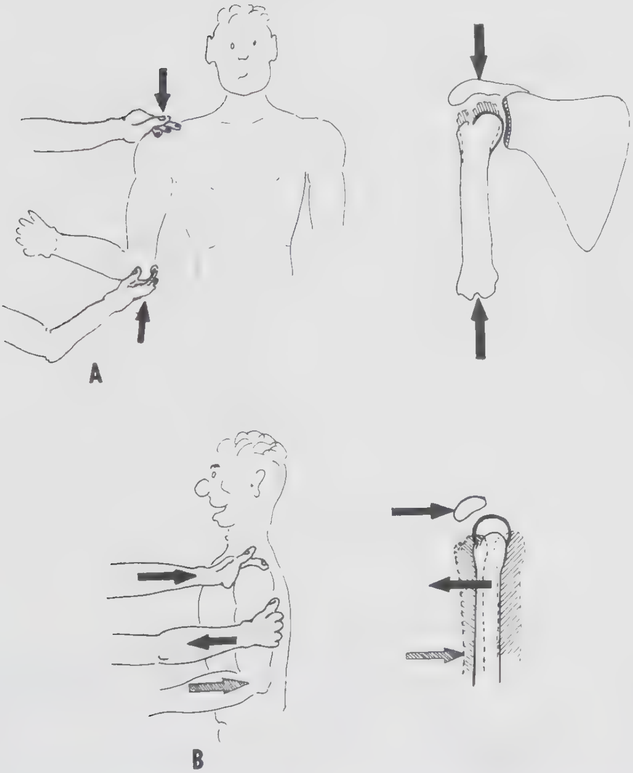


Figure 10–15. Manipulation treatment of *involuntary* motion of the glenohumeral joint. *(A)* Elevation of the head of the humerus against the glenoid fossa. Pressure along the shaft of the humerus, with the other hand preventing elevation of the scapula, causes the humerus to elevate, thus stretching the superior capsule. *(B)* Anterior and posterior motion of the head of the humerus against the glenoid. Three points of contact must be applied. One hand mobilizes the humerus while the other hand *fixes* the scapula. The elbow or forearm is fixed by the therapist's body or elbow.

of the muscles and the skin and osteoporosis. All can be minimized, reversed, or improved by active, active-assisted, and active-resisted exercises.

I must express a word of caution here. Too often a stellate block or an oral course of steroids is prescribed by the physician, but the sequelae of the RSD are overlooked and ultimate full recovery of the patient's hand function is not accomplished.

Many patients with early incipient RSD (shoulder-hand-finger syndrome) will do very well with early recognition and active therapy without sympathetic interruption. Careful monitoring, however, is mandated to ensure gradual recovery and avoidance of chronic residual impairment. Reflex sympathetic dystrophy grades 1 and 2 must be reversed early, while they are still reversible.

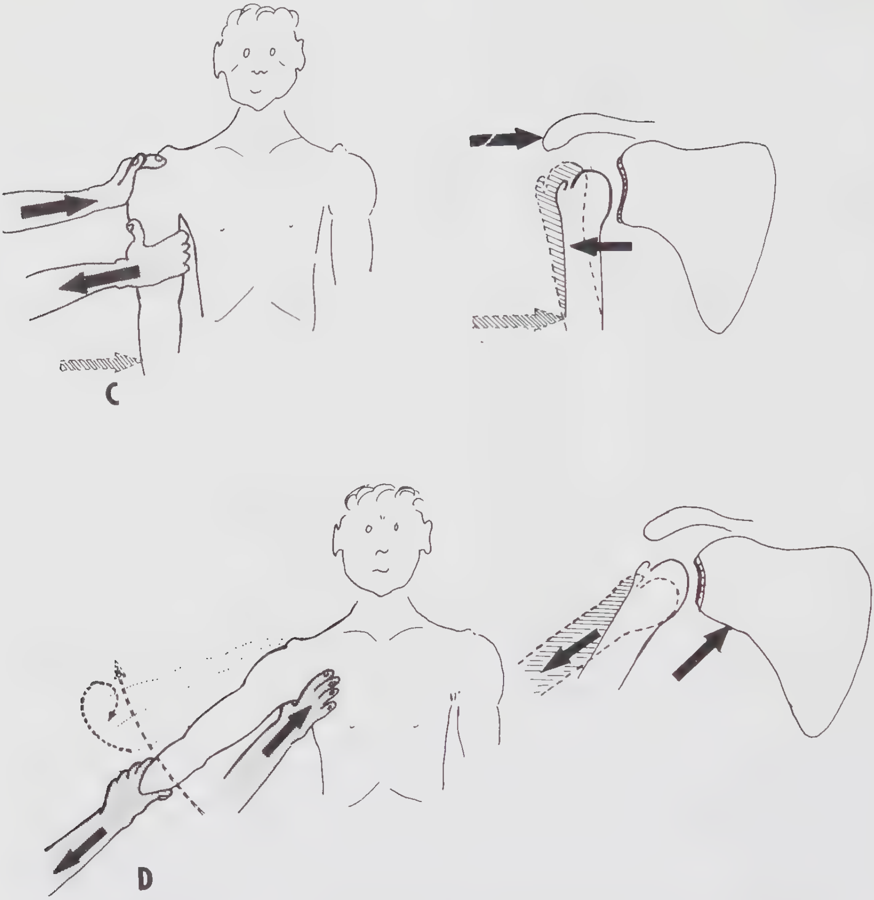


Figure 10–15. (continued). *(C)* Lateral motion of the head of the humerus away (in separation) from the glenoid. One hand of the therapist pulls at a right angle to the shaft of the humerus while the scapula and the elbow are *fixed*. *(D)* Traction to separate the head of the humerus from the glenoid while abducting and gradually externally rotating the arm. Counterresistance (fixation) is applied against the axillary border of the scapula.

Psychiatric Aspects

The full psychiatric implications of RSD are complex, but the subject cannot be dismissed.

The designation of *periarthritic personality* and *type A personalities* are frequently used in medical literature to imply a tendency to develop certain neurologic conditions. The questions of the emotional sides of RSD have been studied by Wirth and Rutherford. The question has been raised as to whether the causalgic state is purely psychogenic and complicated by compensation, hysteria, or even malingering (Shaw).

Given the limited knowledge of the neuro-hormonal-physiologic aspects of many painful states, a relationship can be only suggested with no concrete verification.

It is becoming apparent in neurophysiologic studies that the limbic system is involved in emotional states and that the seratonin cycle is involved in chronic pain. What the effect of serotonin, hypothalamic, or limbic influence is in RSD remains conjectural.

The patient with RSD is indeed under severe duress, and the question as to whether the emotional state is causative or merely a compounding factor is superfluous; both must be addressed. Anxiety must be allayed, and depression must be recognized and addressed. All aspects of treatment, including biofeedback, relaxation therapy, medication, and psychologic counseling must be considered and implemented.

BIBLIOGRAPHY

Benzon, HT, Chomka, CM, and Brunner, EA: Treatment of reflex sympathetic dystrophy with regional intravenous reserpine. Curr Res Anesth 59:500, 1980.

Bonica, JJ: Causalgia and other reflex sympathetic dystrophies. In Bonica, JJ, Liebeskind, JC, and Albe-Fessard, (eds): Advances in Pain Research and Therapy, Vol 3. Raven Press, New York, 1979a, page 141.

Bonica, JJ: The need of a taxonomy. Pain 6:247, 1979b.

Bonica, JJ: Causalgia and other reflex sympathetic dystrophies. In Bonica, JJ: The Management of Pain. Lea & Febiger, Philadelphia, 1990.

Cappen, A: Disorders of mineral metabolism in depressive patients. Psychiatria, Neurologia, Neurochirugia 72:189, 1969.

Christensen, K and Henricksen, O: The reflex sympathetic syndrome: An experimental study of sympathetic reflex control of subcutaneous blood flow in the hand. Scand J Rheumatol 12:263, 1983.

Devor, M: Nerve pathophysiology and mechanisms of pain in causalgia. J Autonom Nerv Syst 7:371, 1983.

Doupe, J, Cullen, CH, and Hange, CG: Posttraumatic pain and the causalgia syndrome. J Neurol Neurosurg Psychiatry 7:33, 1944.

Ecker, A: Norepinephrine in reflex sympathetic dystrophy: An hypothesis. Clin J Pain 5:313, 1989.

Ficat, T, et al: Algodystrophies reflexes post-traumatiques. Rev Chir Orthop 59:401, 1973.

Hannington-Kiff, JG: Relief of causalgia in limbs by regional intravenous guanethidine. Br Med J 2:367, 1979.

Leriche, R: De la causalgie envisagee comme une nevrite du sympathique et son traitment pas la denudation et l'escision des plexus nerveux periarteriees. Presse Med 24:178, 1916.

Letievant, E: Traite de Section Nerveuses. JB Bailliere et fils, Paris, 1873.

Lundberg, U and Frankenhauser, M: Pituitary-adrenal and sympathetic-adrenal correlates of distress and efforts. J Psychosom Res 24:125, 1980.

Meehan, JR: Stress vascular changes and the potential for behavioral modification. J SC Med Assoc Oct, 535, 1963.

Mitchell, SW, Morehouse, GR, and Keen, WW: Gunshot Wounds and Other Injuries of Nerves. JB Lippincott, Philadelphia, 1864.

Mitchell, SW: Injuries of Nerves and Their Consequences. Smith-Elder, London, 1872.

Ochs, S: Axoplasmic transport: A basis for neural pathology. In Dyke, PJ, Thomas, PK, and Lambert, EH (eds): Peripheral Neuropathy. WB Saunders, Philadelphia, 1975, page 213.

Osler, W: Angina Pectoris and Allied States (Lectures). Appleton, New York, 1897.

Owens, JC: Causalgia. Ann Surg 23:636, 1957.

Perroncito, A: La rigenerazione delle fibre nervose. Boll Soc Med Chir Pavia 4:434, 1905.

Rizzi, R, Visentin, M, and Mazzetti, G: Reflex sympathetic dystrophy. In Benedetti, C, et al (eds): Advances in Pain Research and Therapy, Vol 7. Raven Press, New York, 1984.

Roberts, WJ: A hypothesis on the physiological basis for Causalgia and Related Pains. Pain 24:297, 1986.

Sainsbury, P and Gibson, JJ: Symptoms of anxiety and tension and the accompanying changes in muscular system. J Neurol Neurosurg Psychiatry 17:216, 1954.

Shaw, RS: Pathological malingering. N Engl J Med 271:22, 1964.

Shawe, GDH: On the number of branches formed by regenerating nerve fibers. Br J Surg 42:474, 1955.

Steinbrocker, O, Spitzer, N, and Friedman, NH: The shoulder-hand syndrome in reflex dystrophy of the upper extremity. Ann Int Med 29:22, 1948.

Sudeck, P: Über die akute entzundlike knockenatrophie. Arch Klin Chir 62:147, 1900.

Sylvest, J, et al: Reflex dystrophy: Resting blood flow and muscle temperature as diagnostic criteria. Scand J Rehabil Med 9:25, 1977.

Tahmouth, AJ: Causalgia: Redefinition as a clinical pain syndrome. Pain 10:187, 1981.

Wall, PD and Gutnick, M: Ongoing activity in peripheral nerves: The physiology and pharmacology of impulses originating from a neuroma. Exp Neurol 43:580, 1974.

White, JC and Sweet, WH: Pain and the Neurosurgeon: A Forty-Year Experience. Charles C Thomas, Springfield, IL, 1969, page 87.

Wirth, FP and Rutherford, RB: A civilian experience with causalgia. Arch Surg 100:633, 1970.

CHAPTER 11

Scapulocostal Syndrome, Myofascial Pain Syndrome

The reason I discuss scapulocostal syndrome in the same chapter as myofascial pain syndrome is that the offending musculature of each syndrome is identical and the painful sequelae are similar. Only the cause and pathomechanics may differ. Occupational postural stresses are being studied universally as the muscular pain of the upper trapezius region gradually becomes as prevalent as the lumbosacral pain syndrome in industrial *injuries* (Hagberg and associates, 1987). All are fundamentally forms of *shoulder girdle pain.*

SCAPULOCOSTAL SYNDROME

This syndrome, essentially pain in the trapezius muscle group—the levator scapulae and rhomboid muscles—is a painful disabling condition that has been estimated as causing 90 percent of all cases of cervicobrachial pain (Michele and Eisenberg). The condition of fibromyalgia has also gained prominence in recent decades and merits full evaluation. Both are related to the thoracic outlet syndrome, discussed in Chapter 7.

This syndrome is also called *postural fatigue,* and gravity upon the scapular musculature has been considered a cause. The downward rotation of the scapula forces upward and lateral motion of the superior medial aspect of the scapulae (Fig. 11–1) to which are attached these offending muscles. They *fatigue* and become sites of nociception. The postural aspects of scapulocostal syndrome are incurred daily in sitting postures (Fig. 11–2) and standing occupational postures (Fig. 11–3).

In patients with this syndrome there has been no guide to the cause of the

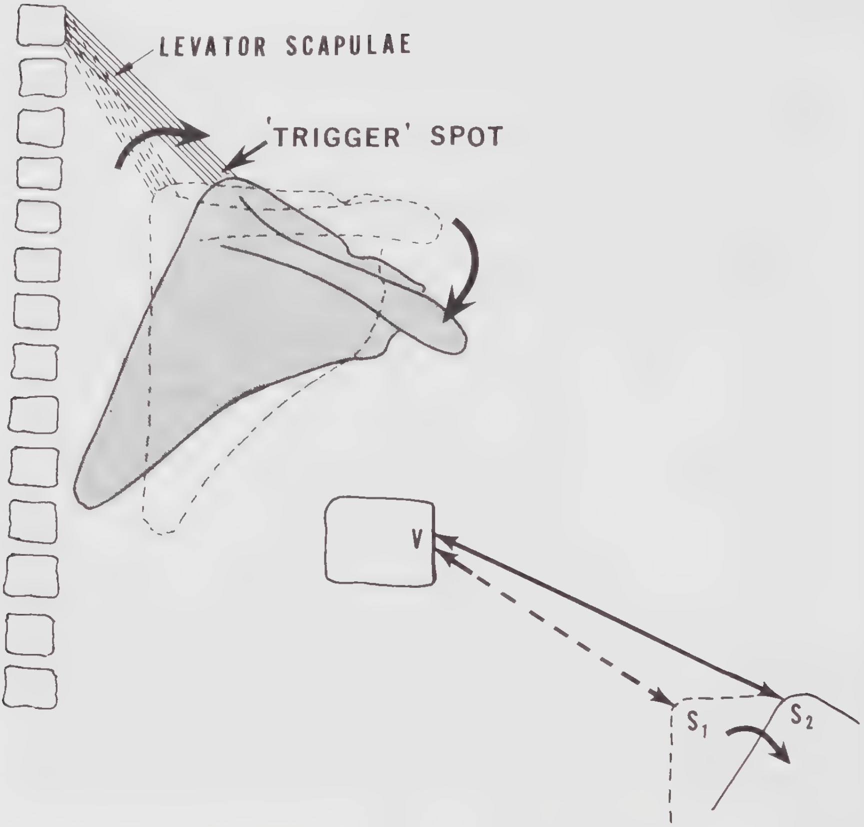


Fig. 11–1. Levator scapulae *trigger* zone in postural fatigue syndrome. With lateral downward rotation of the scapula (*curved arrows*) the superior medial angle of the scapula moves the insertion of the levator scapulae muscle ($V\text{-}S_1$ to $V\text{-}S_2$). The muscle under this traction becomes ischemic, inflamed, and thus tender and painful at the *trigger* site.

disorder, inasmuch as the symptoms are mostly subjective. No signs of inflammation, rheumatic or neuromuscular disease have been elicited. There is local muscular tenderness. Electromyography studies on patients with this syndrome revealed no abnormality except for *fatigue* (decrease of myoelectric amplitude and mean power frequency) on the EMG (Hagberg and Kvarnstrom). This fatigue was not related to any biochemical or metabolite changes or any biopsy findings within the muscle.

The effect of ischemia from sustained muscular isometric contraction has been imputed as a cause of symptoms. With 20 percent of sustained isometric contraction (Edwards) the circulation of a muscle is reduced. Accumulation of lactate also results, causing a low pH which in turn inhibits normal function of

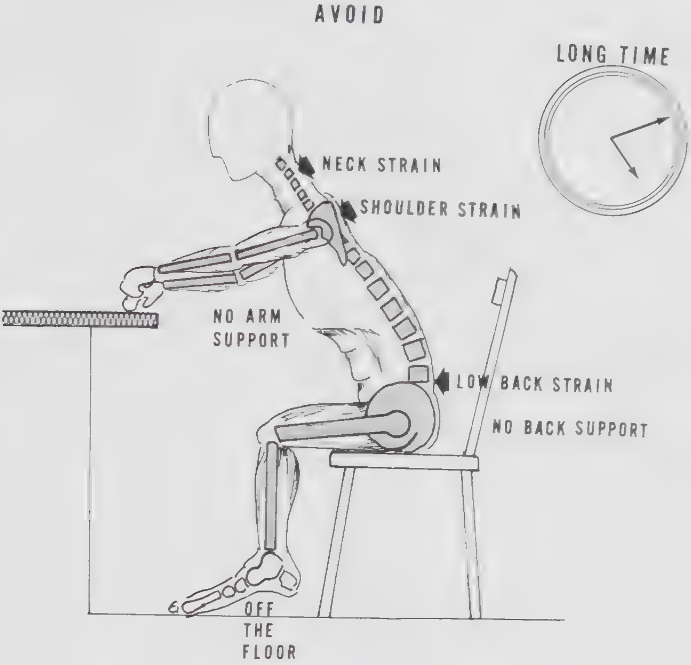


Fig. 11–2. Sitting posture to avoid.

muscle enzymes (Sahlin and associates). This muscular ischemia results in reduction of strength, coordination, and endurance as well as discomfort and pain. Rest for periods of as short as 2 seconds has been shown to alter this metabolite sequence (Hagberg 1981), so ischemia as a cause of prolonged or delayed muscular pain remains improbable, inasmuch as brief rest restores physiologic balance.

Metabolic studies are in progress regarding the elevation of serum creatine phosphokinase (CPK) after muscular exertion with resultant pain and tenderness. Elevation of SCK indicates energy depletion adenosine triphosphate (ATP) of the muscle (Thomson and colleagues), a finding that holds promise for evaluating painful muscle diseases and determining their origins. So far this theory has not been implemented in enough conditions to verify a cause-and-effect relationship.

Holding the arms forward in a sustained position also induces fatigue in the shoulder glenohumeral joints (deltoid) as well as in the supporting scapular musculature (trapezius and serratus). The lever arm of the forward arm-hand position causes the weight of the arms to be very significant in respect to the supporting scapular muscles. The symptoms of fatigue also emit from the tendons, joint capsules, and ligaments as well as from the muscles and their fasciae.

Aching of muscles has been studied after exercise, but little documentation

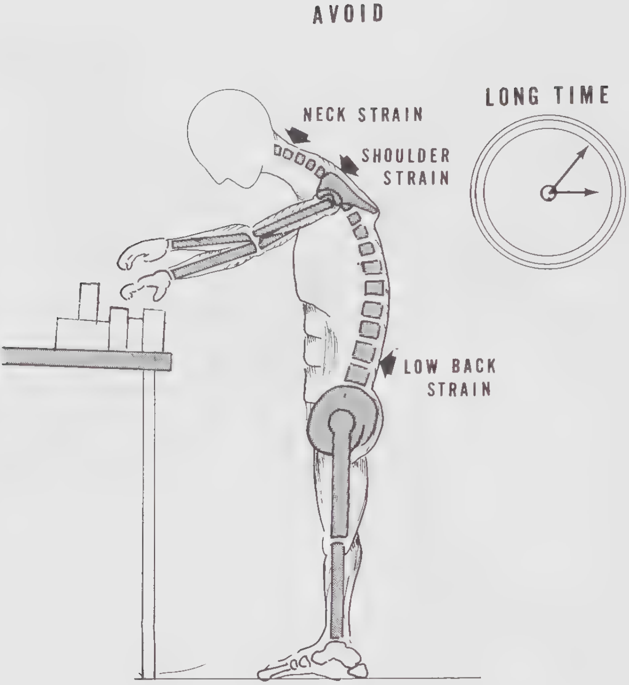


Fig. 11–3. Standing posture to avoid.

has been generated from sustained positions assumed in occupations and activities of daily living. The *overuse syndrome,* in which muscles are painful and deficient after prolonged exercise, is also undergoing study (Dennett and Fry). Some structural and enzymatic changes have been noted in the *overused muscles.* Recently studies of postpoliomyelitis patients have been undertaken to determine whether new disease is responsible rather than overuse of the remaining uneffected nerves and muscles; both have been implicated.

Studies of acute injuries in which the forward-held arm is subjected to an acute stress that causes an acute muscular contraction rather than the sustained isometric muscular contraction of the postural position have revealed ultrastructural muscle ruptures (Friden and colleagues) in the Z structures. The injuries reported, however, were severe and not the usual stresses incurred by postural occupational forces. These ruptures allegedly cause an outflow of metabolites and edema, activating the pain receptors. Injury to collagen also has elevated hydroproline excretion and is considered to be responsible for the reported soreness (Abraham), but, again, these are not the usual occupational postural stresses implicated and need be considered only when the injury is known to be acute and severe.

The ischemia of the supraspinatus tendon has been thoroughly discussed as the so-called ischemic critical zone (Rathburn and MacNab) in earlier chapters. Elevated arm postures requiring sustained contraction of the supraspinatus muscles allegedly cause ischemia within seconds. It is thus apparent that repeated or sustained supraspinatus muscle contraction will initiate a prolonged ischemia of this muscle with ultimate resultant tissue enzymatic changes.

These soft tissue changes, known as *mesenchymal transformation*, have been studied by Fassbender and Wegner. These are possibly the painful degenerative changes that ultimately occur to cause residual persistent pain and impairment. Regardless of the exact vascular, enzymatic, or structural changes that induce the painful shoulder in occupational postural situations, it becomes apparent that a modification of these postures would be reasonable and beneficial. The frequent 1-minute break (Cailliet and Gross) from the task in which the person literally "pulls away from his or her work posture" is scientifically sound. Improvements in sitting and standing postures are also indicated. Strengthening of the affected muscles merits attention, and change of work conditions (ergonomics), such as a change in chair and desk height also warrants attention.

FIBROSITIS AND MUSCULAR REFERRED PAIN SYNDROMES

Chronic musculoskeletal pain syndromes with no identifiable organic causes have perplexed clinicians for centuries. Since 1800 (Simons) the conditions of fibrositis and fibromyalgia have been recognized clinically. They have been termed by various diagnostic labels as their origins varied. The following three categories have evolved (Wilke and Corbo):

1. *Nodular,* in which the muscles are painful and tender due to *foreign matter* deposited in muscles, forming *nodules*
2. *Neurovascular,* in which a form of trauma resulted in a *trigger point*, which, when palpated, activates a distal area of hyperalgia and distal vasodilatation (Travell and Simons)
3. *Psychogenic,* in which all related symptoms are attributed to psychologic deviations and overreaction to normal physiologic changes secondary to trauma

Fibrositis and fibromyalgia (Wilke and Corbo; Wilke and Mackenzie) have been postulated as a disordered pain modulation in which tension, anxiety, and depression adversely affect stage 4 sleep, causing a lowering of the central nervous system amines and a subsequent lowering of the pain threshold with activation of latent tender points in the muscles. These muscles have also probably been sensitized by fatigue, posture, and occupational postures.

Vascular ischemia has played a prominent role in the etiology of fibrositis and fibromyalgia syndromes. Oxygen-sensitive electrodes placed within subcutaneous and muscular tissues reported a lowered tissue oxygenation in patients affected with these syndromes as compared with that in normal control patients (Lund and colleagues). Other studies (Bonafede and associates) revealed a significantly lower blood flow in exercise of these patients when compared with that in normal control patients. The exact mechanism of this hypovascularity remains to be clarified, but it does stress the factor of tissue ischemia as a cause of fibrositis and fibromyalgia: how remains speculative.

Numerous authors have become associated with these syndromes, most notably Smythe and Yanus, who have stated criteria for making a specific diagnosis.

The condition of fibrositis is not exclusively a painful shoulder condition, but the shoulder—that is, the upper trapezius and midline neck region—is a prominent site of pain, thus a discussion of fibrositis appears pertinent in a discussion of the painful shoulder.

Fibrositis is a chronic musculoskeletal disorder that is characterized by generalized aches and pains and multiple tender points with no ascertained organic lesion. By definition patients must have these symptoms for a minimum of 3 months before the diagnosis is accepted. Trigger points—taut tender bands—in muscles have become the major aspect of the diagnosis. Yanus has claimed that 92 percent of patients with this diagnosis have shoulder involvement, and McCain claims 54 percent.

If these bands are diffusely localized, they are termed *primary myofascial pain syndrome* (*PFS*). If these nodules are specifically localized, they are termed *myofascial pain syndromes* (*MPS*). There are many similar syndromes that involve temporomandibular pain syndromes, which are now termed *temporomandibular pain and dysfunction syndrome* (*TMPDS*). *Fibromyalgia* is also commonly used but does not, per se, enjoy a specific classification in the toxonomy of the International Association for the Study of Pain (IASP).

A full thorough evaluation of the *chronic musculoskeletal pain syndromes* (*CMPS*) is not in the purview of this text, but a superficial analysis will furnish the relationship between this complex and the painful shoulder.

The original term *fibrositis* was first offered by Gowers in 1904. The concept, then, was that the clinical condition was inflammatory invasion of subcutaneous and muscular tissue, hence the suffix *itis.* The term has persisted in the literature in spite of the fact that inflammation is no longer considered a tenable pathologic factor. No histologic abnormalities have been recognized in the painful tender *trigger* nodules.

Primary fibromyalgia syndrome is the clinical condition in which the patient presents with multiple tender sites in the musculature but also generalized muscular aching, stiffness, and fatigue left unrestored because of impaired sleep.

The diagnostic criteria proposed by Yanus are as follows:

A. Obligatory
  1. Generalized aches and a painful stiffness in at least three anatomic sites

    for at least 3 months' duration
  2. Absence of traumatic injury, structural rheumatic disease, infectious arthropathy, endocrine-related arthropathy, and abnormal laboratory tests

B. Major
  1. Five or more typical and consistent tender points (of which the shoulder girdle is prominent)

There are many minor criteria, such as fatigue, anxiety, depression, and headache.

Smythe presents criteria for PFS as follows:

1. Widespread aching of more than 3 months' duration
2. Local tenderness at 12 of 14 specific sites
3. Skin-rolling tenderness over the upper scapular region
4. Disturbed sleep, with morning fatigue and stiffness
5. Normal erythrocyte sedimentation rate (ESR), serum glutamic-oxaloacetic transaminase (SGOT), rheumatoid factor test, antinuclear factor (ANF), muscle enzymes, and sacroiliac film studies

The following criteria of MPS are endorsed by Travell and Simons:

1. Local tenderness at one or a few points
2. A distant pattern of referred pain
3. The presence of a taut, palpable band
4. A local twitch response to quick tapping
5. Associated muscular weakness and limited movement

Fibrositic tender points in patients with PFS are areas where palpation elicits extreme pain with withdrawal from the examiner's hand. This withdrawal has been termed the *jump sign.* It is commonly elicited in the midpoint of the upper trapezius muscle.

Muscular tension, attributed to a sustained muscular contraction, has been implicated in fibrositis. There is no important loss of motor units and no conspicuous muscle fiber degeneration noted by EMG studies of patients with clinical fibromyalgia (Zidar), nor is there any detectable electrical activity of the involved muscles. Factors other than muscular tension are considered to be responsible for maintaining the pain in this condition.

Sleep and psychologic disturbances are frequently attributed as being causative of—and certainly associated with—PFS and are well documented in the voluminous literature. Treatment of PFS and MPS has been empirical, varying from oral medication—including antidepressants and nonsteroidal anti-inflammatory medication—and physical therapy with numerous modalities, especially spray and stretch techniques as advocated by Travell and Simons.

Clinical Management

Clinical management of fibrositis is essentially education and counseling of the patients to reassure them that the disease is not ominous, serious in sequelae, and that the symptoms are subjective with good prognosis. The patient must understand that the symptoms are *not* imaginary but that they are not indicative of a *disease;* they are a tissue reaction of subjective discomfort with minimal organic sequelae. Patients should be advised of appropriate palliative therapies to avoid excessive prolonged treatments of dubious value.

Because symptoms are mostly postural and concern limited flexibility *(stiffness)*, attention to improving the posture is indicated. This is evaluated in Chapter 5. Flexibility exercises of the shoulder are recommended, as in adhesive capsulitis. Active exercises are to be encouraged, rather than passive treatment, to make the patient more self-sufficient and less dependent. *Fitness* exercises have been advocated and were suggested by Moldofski and colleagues when they found that *normal* patients could, with induced sleep deprivation, develop myofascial symptoms—*except* those who were physically fit and undergoing active exercise programs. Patients routinely assigned to physical fitness programs improved in their fibromyositis symptoms when compared with control patients (McCain, 1988). Aerobic exercises have produced objective diminution by dolorimeter studies.

Nonsteroidal drugs have a limited effect, but sleep-influencing medications have proven to be of value. Doxepin hydrochloride in doses of 10 to 25 mg 1 hour before bedtime has been effective.

Discussion of this condition has been directed to its prevalence in shoulder girdle symptomatology and not to total consideration of the patient impaired by fibromyalgia. A voluminous literature is emerging in these studies.

BIBLIOGRAPHY

Abraham, WM: Factors in delayed muscle soreness. Med Sci Sports 9:11, 1977.

Bonafede, P, et al: Exercise muscle blood flow in patients with fibrositis: A xenon clearance study. Arthritis and Rheumatism, 30 (Suppl):14, 1987.

Bonica, JJ: Causalgia and other reflex sympathetic dystrophies. Postgrad Med 53:143, 1973.

Cailliet, R: Neck and Arm Pain, ed 3. FA Davis, Philadelphia, 1991.

Cailliet, R and Gross, L: The Rejuvenation Strategy. Doubleday, New York, 1987.

Dalackas, MC: Morphological changes in the muscles of patients with postpoliomyelitis neuromuscular symptoms. Neurology 38:99, 1988.

Dalakas, MC, et al: Late postpoliomyelitis muscular atrophy: Clinical, virologic and immunological Studies. Review of Infectious Diseases Vol 6 (Suppl 2), May–June 5562, 1984.

Dinnett, X and Hunter, JHF: Overuse syndrome: A muscle biopsy study. Lancet 1:905, 1988.

Edwards, RHT, Hill, DK, and McDonnell: Myothermal and intramuscular pressure measurements during isometric contractions of the human quadriceps muscle. J Physiol 224:58, 1972.

Fassbender, HG and Wegner, K: Morphologie und Pathogenese des Weichteilrheumatismus. Z Rheumaforsch 32:355, 1973.

Friden, J, Sjostrom, M, and Ekblom, B: A morphological study of delayed muscle soreness. Experimentia 37:506, 1981.

Fry, HJH: Overuse syndrome in musicians—100 years ago. An historical review. Med J Aust 145:620, 1986.

Gowers, WR: Lumbago: its lesions and analogue. Br Med J 1:117, 1904.

Hagberg, M: Occupational Musculoskeletal Disorders —A New Epidemiological Challenge: Progress in Occupational Epidemiology, edited by C Hogstedt and C Reuterwall. Elsevier, New York, 1988.

Hagberg, M: Muscular endurance and surface electromyography in isometric and dynamic exercise. J Appl Physiol 51:1, 1981.

Hagberg, M: Work load and fatigue in repetitive arm elevation. Ergonomics 24 (7):543, 1981.

Hagberg, M: Local shoulder muscular strain: Symptoms and disorders. J Hum Ergol 11:99, 1982.

Hagberg, M and Kvarnstrom, S: Muscular endurance and electromyographic fatigue in myofascial shoulder pain. Arch Phys Med Rehabil 65:522, 1984.

Hagberg, M and Wegman, DH: Prevalent rates and odds ratios of shoulder-neck diseases in different occupational groups. British Journal of Industrial Medicine, 44:602, 1987.

Lund, N, Bengtsson, A, and Thorborg, P: Muscle tissue oxygen pressure in primary fibromyalgia. Scand J Rheumatol 15:165, 1986.

Aching muscles after exercise. Lancet 1:1123, 1988.

McCain, GA: Role of physical fitness training in the fibrositis/fibromyalgia syndrome. Am J Med (Suppl 3A) 81:73, 1986.

McCain, GA and Scudds, RA: The concept of primary fibromyalgia (fibrositis): Clinical value, relation and significance to other chronic musculoskeletal pain syndromes. Pain 33:273, 1988.

Michele, AA and Eisenberg, J: Scapulocostal syndrome. Arch Phys Med Rehabil 49:383, 1968.

Moldofsky, H, et al: Musculoskeletal symptoms and non-REM sleep disturbances in patients with “fibrositis syndrome” and healthy subjects. Psychosom Med 37:341, 1975.

Rathburn, JB and MacNab, I: The microvascular pattern of the rotator cuff. J Bone Joint Surg 52B:540, 1984.

Semble, EL and Wise, CM: The Fibrositis Syndrome: Part II. Clinical Management Options. Pain Management Nov/Dec: 247, 1988.

Simons, DG: Fibrositis/fibromyalgia: a form of myofascial trigger points. Am J Med 81 (Suppl 3A):93, 1986.

Smythe, HA; “Fibrositis” as a disorder of pain modulation. Clin Rheunm Dis 5:823, 1979.

Smythe, HA and Modolfsky, H: Two contributions to the understanding of the 'fibrositis' syndrome. Bull Rheum Dis 28:928, 1977–1978.

Thomson, WHS, Sweetin, JC, and Hamilton, IJD: ATP and muscle enzyme efflux after physical exertion. Clin Chim Acta 59:241, 1975.

Travell, JG and Simons, DG: Myofascial Pain and Dysfunction: The Trigger Point Manual. Williams & Wilkins, Baltimore, 1983, page 12.

Wilke, WS and Corbo, DB: Fibrositis/fibromyalgia: Causes and treatment. Compr Ther 15 (1):47, 1989.

Wilke, WS and Mackenzie, AH: Proposed pathogenesis of fibrositis. Cleve Clin Q 52:147, 1985.

Yanus, MB: Fibromyalgia syndrome: A need for uniform classification. J Rheumatol 10:841, 1983.

Zidar, J, et al: Quantitative EMG and muscle tension in painful muscles in fibromyalgia. Pain 40:249, 1990.

CHAPTER 12

Visceral Referred Pain

The exact mechanism of visceral referred pain has been under study for many decades, and there remains speculation as to its exact neurologic mechanism. Clinically, however, the existence of referred pain from a hollow viscera has been validated. The hollow viscera has varied from cardiac blood vessels (angina), gallbladder, and intestine, and the sites of referral have been to the shoulder, scapular area, anterior chest wall, and so forth. The area of referred pain has also been coupled with areas of hypersensitivity of the overlying skin (Fig. 12–1).

The postulated mechanisms of referred visceral pain cannot be thoroughly discussed here but have been summarized by Bonica. It is postulated that sensory impulses from visceral organs bombard the central nervous system, setting up an *irritable focus* within the cord in the region of the substantia gelatinosum (SG in Fig. 12–2).

Impulses from the *diseased* viscus provoke impulses that enter the irritable focus by way of afferent fibers through the dorsal root ganglia (DRG). Two pathways are initiated by an internuncial neuronal connection. One goes to the contralateral portion of the cord and ascends to the thalamus and hypothalamus to record the sensation of pain in the cerebral cortex. A branch stimulates the anterior horn cell (AHC), which causes muscular contraction (*spasm*), which in turn sends sensory impulses back to the cord by way of dorsal root ganglia to enhance the pain cycle.

Another pathway has been postulated for referred skin pain by way of afferent impulses from the viscus to the skin, then to the dorsal root ganglia paralleling the afferent fibers from the viscus (dotted line in Fig. 12–2).

Tissue damage from the ischemic cardium following an infarct also may stimulate vagal and sympathetic afferent impulses that transmit pain through the afferent fibers to the dorsal ganglia, then the substantia gelatinosum.

The diaphragm is considered to be the most frequent site whereby extrinsic

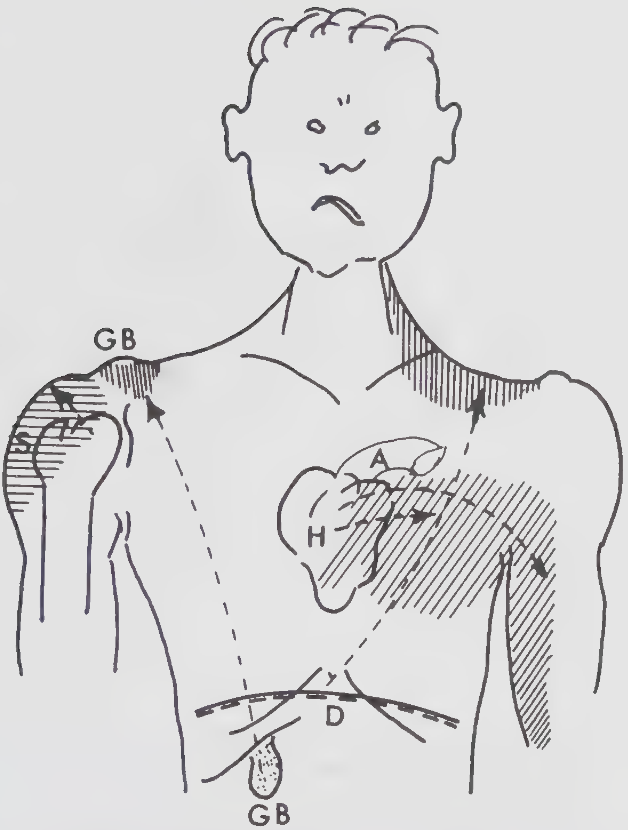


Fig. 12–1. Visceral sources of *shoulder* pain. The word *shoulder* is used vaguely in referred pain from visceral sites. Pain that originates in the shoulder is localized directly over the glenohumeral area (S). Irritation of the diaphragm (D) causes referred pain in the trapezius area. Myocardial origin (H and A) refers pain to the axilla and left pectoral region. Gallbladder irritation (GB) refers pain to the tip of the shoulder and posteriorly in the scapular region.

viscus disease refers pain to the shoulder. This pain is mediated by the phrenic nerve, which is formed by a root of the anterior primary division of the C_4 root; thus, its afferent fibers originate usually in the third and fourth cervical segments (constantly from C_4 and variably from C_3 and C_5).

Each phrenic nerve receives gray rami communications from the superior and middle cervical sympathetic ganglia, hence the nerve is accompanied by sympathetic fibers that transmit sensory impulses from the diaphragm. Because of their origin in the cervical roots that also innervate the shoulder region (C_4 to C_5), it is neurologically apparent that somatic and sympathetic nociceptor impulses can be transmitted to the shoulder region from irritation of the diaphragm.

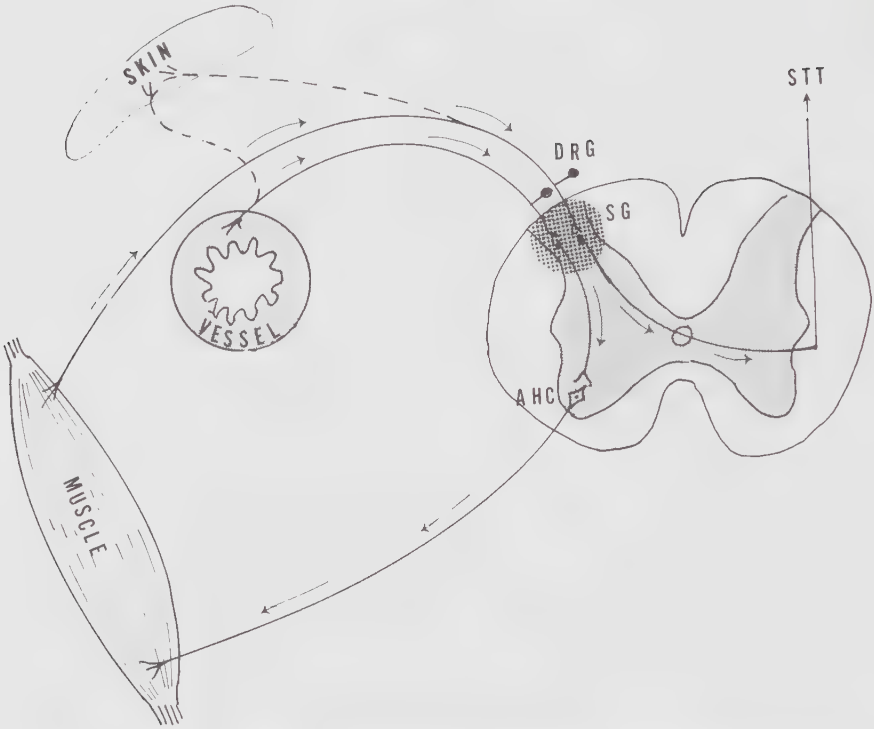


Fig. 12–2. Neural mechanism of visceral referred pain. When there is nociception of a viscus (for example, coronary, gallbladder, intestine), afferent impulses go to the substantia gelatinosum (SG), which may become an *irritable focus* from repeated barrages of nociceptor impulses. Then there can be one or two transmissions: (1) by way of the spinal thalamic tracts (STT) to the hypothalamus then the cortex, or (2) by way of an internuncial tract to the anterior horn cell (AHC), which causes the muscle to contract. The muscle contraction may become an afferent nociceptor impulse through its dorsal root ganglion (DRG).

A circuitous pathway (dotted line) depicts nociceptor impulses that go to the skin, which in turn sends afferent impulses through the DRG into the SG. The skin becomes hypersensitive, and the muscle undergoes *spasm*. Both of these effects are the clinical evidence of referred visceral pain; that is, the shoulder pain of a coronary attack or gallbladder attack.

The phrenic nerve has many sites of possible irritation as it descends vertically in front of the anterior scalene muscle, then through the superior thoracic outlet within the mediastinum, along the pericardium, and finally to innervate the superior and inferior surfaces of the central diaphragm. Irritation of the diaphragm refers pain to the supraclavicular region, the trapezius muscle, and the superior angle of the scapula (Capps and Coleman; Head; Lelan).

Any pain in an isolated region, such as the shoulder, always presents the possibility of a significant organic distal lesion and must not be diagnosed exclusively as pain from that musculoskeletal area. A pain in the shoulder may be of a cardiac, diaphragmatic, or upper abdominal viscus origin. The shoulder pain will disappear after attention is paid to the basic viscus lesion and thus has been instrumental in making this latter diagnosis.

BIBLIOGRAPHY

Bonica, JJ: The Management of Pain, Vol 1, ed 2. Lea & Febiger, Philadelphia, 1990, page 170.

Capps, JA and Coleman, GH: Experimental and Clinical Study of Pain in Pleura, Pericardium and Peritoneum. Macmillan, New York, 1932.

Head, H: On disturbances of sensation with especial reference to the pain of visceral disease. Brain 16:1, 1893.

Lelan, JL: Visceral aspects of shoulder pain. Bull Hosp Joint Dis Orthop Inst 14:71, 1953.

INDEX

Abdominal viscus, pain from, referred to shoulder, 262–265
Acromial process, functional anatomy of, 3, 4f, 10, 17, 39f
Acromioclavicular disk, development of, 38, 39f
Acromioclavicular joint
  functional anatomy of, 2f, 2–3, 9–10, 34, 38–40, 41f, 55f
  injury, 144–147
    diagnosis, 144–145
    immobilization of arm with, 145–147, 146f
    mechanisms, 144
    treatment, 145–147
  motion, 38–40
  osteoarthritis, 90–91
  palpable trigger point, 70f
  as site of pain or malfunction, 53, 53f
Acromioclavicular ligaments, 38
Acromiocoracoid arch, functional anatomy of, 12f, 39f
Acromionectomy, 102f, 102–103
Acromium
  in acromioclavicular joint injury, 144–145
  functional anatomy of, 16, 28, 30, 38, 55f, 124
  osteoarthritic spurring of, 109
Acupuncture, for causalgic pain, 245
Adhesive bursitis, 76. *See also* Frozen shoulder
  formation of, 66, 67f
Adhesive capsulitis, 15, 76, 113–116. *See also* Frozen shoulder
  radiologic signs of, 112, 113f
Adson test, in scalene anticus syndrome, 183–184, 184f
Aging
  changes in, 149
  effects on glenohumeral joint, 56–57
Air-inflated splint, 207, 208f
Alpha fiber(s), 19
Analgesic agent, intra-articular injection of, 116
Anesthetic agents, local injection of, 79–81, 80f
Ankylosing spondylitis, 93
Anterior circumflex artery, 25, 27f
Anterior horn cells, 21, 21f, 262, 264f
Anxiety, and tendon reflexes, 22
Apprehension test, 155, 156f
Arachidonic acid metabolites, 79
Arm. *See also* Upper extremity
  immobilization. *See* Immobilization
  movement, 42–46
    abduction, 43, 47f–48f, 124
    painful arc of, 62–63, 71, 74f
    planes of, 27, 29f, 29–30, 138, 140f
    rotation, at glenohumeral joint, 14, 14f
Arthritis, posttraumatic, 145
Arthrography
  in adhesive capsulitis, 112, 113f
  of rotator cuff tears, 99, 101f
Arthropathy, 223
Arthrotomy, for frozen shoulder, 119–120
Astereognosis, in hemiplegia, 217, 218f
Athletes. *See also* Sports injuries
  occult shoulder subluxation in, 136–137
Axillary blood vessel injury, 188
Axillary nerve, 23, 26
Axoplasmic neural transport, 229f, 229–230

Beta efferent fibers, 22f
Biceps brachii muscle, functional anatomy of, 47, 49f
Biceps mechanism, functional anatomy of, 3–4, 13, 46–50
Biceps muscle
  function of, 47–48

    in hemiplegia, 214
    strengthening exercise, 167
Biceps tendinitis, 108, 114–116
Biceps tendon(s)
    degeneration of, 109
    effects of aging on, 57
    entrapment, 154
    functional anatomy of, 13, 13f, 18, 24, 24f, 47–50, 49f, 55f
    in hemiplegia, 214
Bicipital groove
    palpable trigger point, 70f
    as site of pain or malfunction, 53f
Bicipital tendinitis, 50
Bicipital tendon, as site of pain or malfunction, 53, 53f
Biofeedback, in treatment of hemiplegia, 219, 221
Bowling, hyperextension for, 156, 158f
Brachial plexus, 169, 170f, 177, 179f, 188f
    compression or angulation, in thoracic outlet syndrome, 181–182
Brachial plexus injuries, 186–189
    with hemiplegic shoulder, 212
    mechanisms of, 186
    neurologic examination of, 187
Brachial plexus neuritis, 188–189
Brachial plexus neuropathy, 188
Brachial radiculitis, acute, 188
Brisement treatment
    for frozen shoulder, 122
    of rotator cuff tear, 99, 101f
Burning pain, 227–229. *See also* Reflex sympathetic dystrophy
Bursitis. *See also* Subdeltoid (subacromial) bursitis
    formation of, 66, 67f

Calcific tendinitis, 63–66
    development of, 63–65, 65f
    radiologic diagnostic tests, 71–73
    symptoms of, 65–66
Calcium boil, 66, 67f
Cardiac pain, referred to shoulder, 262–265
Causalgia, 227–228
    treatment, 243–245
Central nervous system, anatomy, 194f
Cerebrovascular incident. *See also* Stroke
    flaccid stage, 194–195
        shoulder in
            pathomechanics of, 201–202
            treatment, 207–209, 208f, 210f–212f
    functional evaluation of patient after, 222–223
    perceptual deficits after, 221–222
    sensory deficits after, 221
    spastic stage, 195–197
        complete, 204
        incomplete, 204
        partial, 204
        sensory involvement in, 217–218
        shoulder in
            pathomechanics of, 202–205
            treatment, 214–223
        synergy patterns in, 215–217
    spontaneous recovery from, 194–196, 195f
    stages of, 193–198
        shoulder in, 198–201
            pathomechanics of, 201–205
    synergy stage, 198
        shoulder in, pathomechanics of, 205
    transient ischemic attack stage, 193–194
Cervical discogenic disease, 174–175
Cervical dorsal outlet syndrome. *See* Thoracic outlet syndrome
Cervical lordosis, development of, 127–128
Cervical nerve, fibers, 168–169, 169f
Cervical nerve root involvement, 168–172
    dermatomal/myotomal symptoms of, 171f–175f, 171–172
    diagnostic tests, 175, 183–184
    eighth cervical nerve root irritation, 172f
    examination for, 173–175, 177f–178f
    imaging with, 175
    pain from, 171–172, 174f
        mechanism of, 172–175
    seventh cervical nerve root irritation, 172f
    sixth cervical nerve root irritation, 171f
Cervical outlet, definition of, 182
Cervical rib(s), 177, 182
Cervical spine
    foramina, 168, 169f
        opening, variations in, 173–175, 176f
    foraminal grooves, 168–169, 170f
    functional unit of, 168, 169f
Chemical mediation, of chemical nociceptors from trauma, 58, 61f
Chronic musculoskeletal pain syndromes, 258
Clavicle
    functional anatomy of, 3, 38–39, 179f
    motion of, 40, 41f–42f
    muscles acting on, 41–42, 44f
Clavicular fractures, 147
Claviculocostal syndrome, 183
    diagnostic tests, 184, 185f
Claviculoscapular ligaments, functional anatomy of, 40, 41f
Codman exercises, 76, 77f
Collagen fiber intersectional gliding and adhesion, 75, 75f
Collagen fibers, of tendons, 91–92
Compression notch of Hermodsson, 140, 141f
Conjoined tendon
    functional anatomy of, 10, 23, 24f
    as site of pain or malfunction, 53, 53f
    tendinitis of, 50

Conoid ligament, functional anatomy of, 39–40, 40f–41f
Contrast baths, 246–247
Coracoacromial ligament, functional anatomy of, 3, 4f, 11, 12f, 17, 28–30, 39f, 55f
Coracoclavicular ligament, functional anatomy of, 11, 39, 39f–40f
Coracohumeral ligament, functional anatomy of, 11, 11f, 15, 24, 50
Coracoid process, functional anatomy of, 3, 4f, 10f, 10–11, 17, 39f, 47
Costoclavicular compression syndrome, 183
  diagnostic tests, 184, 185f
Costoclavicular maneuver, 184, 185f
Costosternal joint, as site of pain or malfunction, 53, 53f
Costovertebral joint(s)
  functional anatomy of, 2f, 2–3, 33f
  as site of pain or malfunction, 53, 53f
Creatine phosphokinase, in postural fatigue syndrome, 255
Critical zone, 54, 256–257
  definition of, 25
  degenerative thinning and fissuring of, 95, 96f
  functional anatomy of, 25–26, 26f–27f

Degenerative acromioclavicular arthritis, 114–116
Deltoid muscle
  abduction angle of, 28, 32f
  action of, 28, 28f, 31f
    on glenohumeral joint, 44, 46f
  degeneration, impairment, fatigue, or pain, 54
  functional anatomy of, 18, 26–27, 28f, 41–42, 44f, 55f, 200–201
  rest length, 44
  strengthening exercises for, 86, 167
Denial, in hemiplegia, 217, 218f
Dermatomes, 169
Diaphragm, as site of visceral referred pain transmission, 262–263
Dips exercises, for hyperextension of shoulder, 156, 160f
Distention arthrography, for frozen shoulder, 122
Dorsal root ganglia, 21f, 262, 264f
Dorsal scapular nerve, 36
  entrapment, 191
Drop arm test, positive, 99, 100f
Drop test, 175
Dystrophy, definition of, 228

Edematous extremity. *See also* Shoulder-hand-finger syndrome, hand edema in
  antigravity treatment of, 119f
Elbow flexion, 47–48
Emotions, effect on posture, 131–133
Erb's palsy, 187
Erector spinae muscles, 201
Exercise(s). *See also* Pendular exercise
  Codman, 76, 77f
  Dips, for hyperextension of shoulder, 156, 160f
  flexibility, 157, 162f–165f
    for thoracic outlet syndrome, 186, 187f
  for frozen shoulder, 121, 121f
  hyperextension, 156, 159f
  physiologic effect of, 87–93
  posture, 131, 133f
  posture-scapular elevation, 187f
  rehabilitation, 164–166
  side bending, 164f–165f
  and sports injuries, 150
  strengthening
    for biceps muscle, 167
    for deltoid muscle, 86, 167
    for pectoralis major muscle, 167
    for rotator cuff, 84–87, 90f–91f, 166f, 167
    for shoulder girdle muscles, 84–87, 90f–91f
  stretching, to increase range of motion, 155, 157f
  swinging warm-up, 157, 162f
  trunk twist, 163f
Extrafusal spindle system, 18–22, 22f

Fascial remnants, 177
Fatigue. *See* Postural fatigue syndrome
Fibromyalgia, 253, 257–260
Fibrositis, 257–260
  definition of, 258
  management, 260
Fingers. *See also* Shoulder-hand-finger syndrome
  joints, mobilization, 247, 248f
First rib, 40–41, 43f
Flexibility, total body, 157
Flexibility exercises, 157, 162f–165f
  for thoracic outlet syndrome, 186, 187f
Foramen of Weitbrecht, 15, 15f, 135, 136f
Forearm, supination of, 47–48
Forearm drop test, for rotator cuff tears, 99, 101f
Forward-head posture, 131, 132f
Fracture
  clavicular, 147
  interligamentous, 147
  intra-articular, in shoulder dislocation, 139–140, 141f
  Neer type II, 147
Frozen shoulder, 15, 79, 102, 105–122

  active and passive exercises for, 121, 121f
  active range of motion in, limited, 110
  arthrotomy for, 119–120
  benign neglect of, 112
  brisement for, 122
  clinical picture in, 110–112
  conditions relating to, 113–116
  definition of, 105
  distention arthrography for, 122
  home exercises for, 120f
  manipulation technique for, 121–122
  outcome, 117
  physical findings at surgery, 118–119
  physical findings in, 110–112
  radiologic signs of, 112
  slow continuous traction for, 120–121
  stages of, 106, 107f, 108–109
  surgical treatment, 117–118
  tissues involved in, 105–109
  tissue site of pain in, 110
  treatment of, 112–121

Gamma efferent fibers, 21–22, 22f
Glenohumeral joint
  active pendular exercise, 76–77, 78f
  capsule
    action during movement, 13–14, 14f
    anterior, 15, 15f
    functional anatomy of, 12–16, 13f
    pouch in, 15, 15f
    as site of pain or malfunction, 53f, 54
  deltoid muscle action on, 44, 46f
  dynamic stabilizers, 150
  functional anatomy of, 1–3, 2f, 6–11, 150
  gliding motion at, 14, 14f, 31f
  immobilization, 75–76
  joint play of, 15, 16f–17f
  kinetic muscular functions in, 18
  manipulation treatment of involuntary motion of, 16f–17f, 114–115, 115f–116f, 247, 249f–250f
  microtrauma, 149–150
  mobilization, 247, 249f–250f
  motion of, 27–33
  musculature of, 18
  palpable trigger point, 70f
  passive range of motion, 15, 16f–17f
  passive range of motion exercises. *See* Codman exercises
  range of motion, postural effect on, 63, 64f
  rhythmic stabilization manipulation of, 115, 117f–118f
  as site of pain or malfunction, 53, 53f, 54
  sites of adhesion formation in, 105–106, 106f
  socket of, 12f
  static muscular functions in (static stabilizers), 18, 150
  subluxation, in hemiplegia, 201, 203f, 209–212
    diagnosis, 210–211
    prevention, 210, 213f
    treatment, 211–212, 213f–215f
  tuberosities, wearing away of, 56–57, 57f
Glenohumeral ligaments, 14–15, 15f, 135
  functional anatomy, 150
Glenoid fossa
  functional anatomy of, 9, 10f, 11–13, 12f–13f, 17, 22, 23f, 36, 39f, 47, 124, 198–200, 199f
  in hemiplegia, 201, 203f
Glenoid labrum
  functional anatomy of, 55f, 150
  in shoulder dislocation, 135–136
Global shoulder pain, in pitcher or tennis player, 154
Golgi system, 19
Greater tuberosity
  functional anatomy of, 29–30, 33f, 55f
  impingement on acromial process, 124–126, 125f
  palpable trigger point, 70f
  as site of pain or malfunction, 53f
  tenderness over, 110
Guanethidine, intravenous, for causalgic pain, 245

Hand. *See also* Shoulder-hand-finger syndrome
  clawlike, in shoulder-hand-finger syndrome, 242
  intrinsic minus, in shoulder-hand-finger syndrome, 241, 242f
Head, musculature of, 178–179, 180f
Heat therapy, 75, 77, 114
Hemiplegia
  shoulder in, 198–201
  treatment
    Bobaths' concept of, 217–219
    muscle reeducation in, 219–221
    proprioception neuromuscular facilitation in, 217, 220, 222f
Hemiplegic patient. *See also* Cerebrovascular incident; Stroke
  stages of stroke in, 193–198
Heterotropic ossification, in stroke patient, 223–224
  treatment, 224
Humerus, 27
  abduction of, 31f–32f, 45
  external rotation of, 33f, 45
  functional anatomy of, 55f, 200
  in hemiplegia, 201, 203f, 214
  internal rotators of, in hemiplegia, 203–204, 204f
  motion of. *See also* Scapulohumeral rhythm, humeral phase of

    - on biceps tendon, 47–48, 49f
  - sites of muscle origin and insertion upon, 19f
- Hyoideus muscle, 180f
- Hyperabduction compression syndrome, 183
- Hyperextension
  - for bowling, 156, 158f
  - Dips exercises for, 156, 160f
  - exercise for, 156, 159f
- Hyperextension theory, of shoulder dislocation, 138–139, 139f

- Ice therapy, 77, 114
  - for glenohumeral joint, 73–75
  - for inflammation, 79
  - in sports injuries, 161
- Imaging. *See also* Magnetic resonance imaging (MRI)
  - with cervical nerve root involvement, 175
  - of glenohumeral subluxation, 155
  - with shoulder dysfunction, 56, 57f
- Immobilization
  - with acromioclavicular joint injury, 145–147, 146f
  - of glenohumeral joint, 75–76
  - with shoulder dislocation, 142, 143f
  - in sports injuries, 160
- Impingement syndrome, 59–60
- Industrial injuries, 253
- Inflammation, in sports injuries, 159–160
- Infraspinatus fossa, 10f
- Infraspinatus muscle
  - action of, 29, 31f
  - calcium deposits in, 66, 71
  - functional anatomy of, 10, 10f, 18, 20f, 23, 24f
  - function of, 32f, 45
  - in hemiplegia, 203
- Intrafusal spindle system, 18–22, 22f
- Intrinsic minus hand, 241, 242f
- Ischemia, in fibrositis and fibromyalgia, 257–258

- Joint
  - abnormal, 68–69
  - arc-slide, 6, 7f
  - definition of, 3
  - hydrodynamic lubrication, 5, 6f
  - incongruous, 5f, 6–8, 9f
    - motion of, 6, 7f
    - muscular action of, 8, 9f
  - mechanics of, 4–6
  - normal, normal stress on, 68–69
  - rotation, 6, 7f, 9f
  - spin, 6, 7f
  - spin rotation, 6, 7f
  - swing, definition of, 7f
  - true congruous, 5–6
    - definition of, 4–5
    - motion of, 5–6, 8, 9f
- Joint capsule, 4
  - function as ligament, 6, 8f
- Joint play
  - definition of, 15
  - of glenohumeral joint, 15, 16f–17f
- Joint surface(s), 4, 5f
  - asymmetrical, 5, 5f
  - ovoid, 4, 5f
  - sellar, 4, 5f
- Jump sign, 259

- Klumpke's paralysis, 187
- Kocher manipulation, for closed treatment of dislocation, 142, 143f

- Latissimus dorsi muscle
  - functional anatomy of, 34, 36–37, 37f
  - in hemiplegia, 203–204, 204f, 214
- Lesser tuberosity
  - functional anatomy of, 33f
  - palpable trigger point, 70f
  - as site of pain or malfunction, 53f
- Leukotrienes, 79
- Levator scapulae muscle(s)
  - functional anatomy of, 34, 36, 37f
  - function of, 45–46
  - in postural fatigue syndrome, 253, 254f
  - trigger zone, 254f
- Ligament(s), 6, 8f. *See also specific ligament*
- Localized nontraumatic neuropathy, 188
- Longissimus capitis muscle, 180f
- Longissimus cervicis muscle, 180f
- Longus capitis muscle, 180f
- Luxatio erecta, 142

- Magnetic resonance imaging (MRI), of rotator cuff tears, 99
- Manipulation technique, for frozen shoulder, 121–122
- Manipulation treatment, of involuntary motion of glenohumeral joint, 16f–17f, 114–115, 115f–116f, 247, 249f–250f
- Manubrium sternum, functional anatomy of, 40–41, 43f
- Medulla, 195, 196f
- Mesenchymal transformation, 257
- Metacarpal joint, mobilization, 247, 248f
- Metacarpophalangeal joint(s)
  - flexion-extension of, 239–240, 240f
  - mobilization, 247, 248f
- Midbrain, 195–197, 196f
- Moth-eaten fibers, 93

Muscle(s)
  of head and neck, 178–179, 180f
  overuse, 155
  rehabilitation exercise(s), 164–166
Muscle fiber(s), 19
Muscle reeducation, in hemiplegia treatment, 219–221
Muscle spindle system, 18–22
Musculoskeletal pain syndrome(s), causes, 68
Myofascial pain syndrome, 253
  criteria for, 259
  definition of, 258
  primary, 258–260
    criteria for, 258–259
Myositis, 223
Myotomes, 169

Neck, musculature of, 178–179, 180f
Neck reflexes, in hemiplegia, 214–215, 216f
Neer type II fracture, 147
Neuralgic amyotrophy, 188
Neuritis
  acute shoulder, 188
  localized, of shoulder, 188
  paralytic brachial, 188
  serum, 188
Neuroma, formation, 230, 231f
Nodules, 257–258
Nonsteroidal anti-inflammatory drugs, 114
  for inflammation, 79
Nuclear bag, definition of, 19
Nuclear chain fiber, 19, 22f

Obliquus capitis inferior muscle, 180f
Obliquus capitis superior muscle, 180f
Occupational disorders, 93
Occupational postural stresses, 253
Ossification, in stroke patient, 223–224
Osteoarthritis, 89–91
Osteoporosis, in shoulder-hand-finger syndrome, 241–242
Overuse syndrome, 256

Pain. *See also* Burning pain
  of brachial plexus neuritis, 188–189
  causalgia (autonomic) transmission of, 230, 233f
  from cervical radiculitis, 171–172, 174f
    mechanism of, 172–175
  neuron pathways of, 230, 232f
    in visceral referred pain, 262, 264f
  from shoulder-hand-finger syndrome, 236
  of sports injury, management, 159–160
  sympathetically maintained, 228
    neurophysiological mechanism of, 231–233, 234f
  sympathetic independent, 228
Painful arc, of arm motion, 62–63, 71, 74f
Paraspinous muscles, in hemiplegia, 204–205, 205f
Paresthesias, 171
Pectoralis major muscle
  functional anatomy of, 11, 13f, 34, 36–37, 37f–38f, 41–42, 44f
  function of, 46
  in hemiplegia, 203–204, 204f, 214, 217
  strengthening exercise, 167
Pectoralis minor muscle
  functional anatomy of, 11
  in hemiplegia, 203–204, 204f, 214
Pectoralis minor syndrome, test for, 184–185, 185f
Pendular exercise
  active, 76–77, 78f, 81–82, 83f–89f
  passive, 76–77, 77f, 81–82
  for recovery after surgery of rotator cuff tear, 103
Periarthritic personality, 250
Periarthritis, 106
Peripheral nervous system, in reflex sympathetic dystrophy, 230, 231f–232f
Phrenic nerve, in visceral referred pain, 263–265
Pitching
  injuries in, 154–155
  motion of
    acceleration stage, 154
    cocking, 150, 152f, 152–153
    follow-through stage, 154
    late cocking, 150–154, 153f
    windup, 150
  overhead, mechanics and tissues involved in, 150
Pons, 195, 196f
Postural fatigue, 253–257
Postural occupational disease, 55, 58, 59f–60f
Posture, 124–133
  adult, factors determining, 129–130
  and arm function, 46, 124–126
  definition of, 126
  development of, 127–130
  effect on glenohumeral range of motion, 63, 64f
  factors affecting, 126
  familial, 130
  and fibrositis management, 260
  forward-head, 131, 132f
  neurologic concept of, 128, 129f
  orthopedic and neurologic effects of, 126–127
  proper erect, feeling of, 130

- psychologic aspect of, 131–133
- rounded shoulder (dorsal kyphotic), effects of, 57–58, 58f
  - on glenohumeral range of motion, 63, 64f
  - on shoulder anatomy, 124–126, 125f
- sitting, undesirable, 255f
- standing, undesirable, 256f
- and thoracic outlet syndrome, 186

Posture exercise, 131, 133f
Posture-scapular elevation exercises, 187f
Premotor cortex, 196, 197f–198f
Prevertebral fascia, 178–179, 181f
Proprioception, 128–130
Psychogenic disorders, 257

Radiculopathy, from cervical nerve root involvement, 168–172
Range of motion
- for bowling, 156, 158f
- restoration, with reflex sympathetic dystrophy, 245
- and sports injuries, 150
- stretching exercise to increase, 155, 157f

Rectus capitis major muscle, 180f
Rectus capitis minor muscle, 180f
Referred pain, 114–116, 168–191. *See also* Thoracic outlet syndrome
- from cervical radiculitis, 171–172, 174f
  - mechanism of, 172–175
- muscular, 257–260
- visceral-somatic, 262–265
  - neural mechanism of, 262, 264f
  - sites and causes of, 52–53

Reflex, definition of, 229
Reflex sympathetic dystrophy, 227–251. *See also* Shoulder-hand-finger syndrome
- central mechanisms in, 230, 232f–233f
- chemical stellate block for, 243–245, 244f
- definition of, 227
- major, 227
- mechanisms of, 229–230
- mechanoreceptors in, 231–233, 234f
- minor, 227
- minor touch or movement in, pain intensification through, 231–233, 234f
- neurophysiological mechanism of, 231–233, 234f
- pain in, 227–229
- peripheral nervous system in, 230, 231f–232f
- psychiatric aspects of, 230–231, 249–251
- in shoulder lesions, 235–251
- site of, 229
- sympathetic intervention in, 243–245, 244f
- terminology, 228–229
- treatment, 243–249

Rehabilitation exercise(s), 164–166
Relocation test, 155, 156f
Reserpine, intravenous, for causalgic pain, 245
Rest length, definition of, 44
Rheumatoid arthritis, 93
Rheumatoid disease, 93
Rhomboid muscle(s)
- functional anatomy of, 34, 35f, 36
- function of, 45–46
- in hemiplegia, 204–205, 205f, 214

Rhythmic stabilization, 220, 222f
- manipulation of glenohumeral joint, 115, 117f–118f

Rib(s). *See also* First rib
- first thoracic, abnormalities of, 177, 182
- supernumerary. *See* Cervical rib(s)

Rib cage, 9
Righting reflexes, 128, 129f, 201
Rood sling, for treatment of shoulder subluxation with hemiplegic shoulder, 212, 213f
Rotator cuff
- action of, 28, 31, 32f–33f
- circulation to, 25–26, 26f–27f
- degeneration, impairment, fatigue, or pain, 56
- eccentric (deceleration) contraction, 154
- entrapment of, 126
- functional anatomy of, 18, 22–24, 24f, 55f
- inflammation, therapy for, 79
- strengthening exercises for, 84–87, 90f–91f, 166f, 167
- tears, 56, 93, 95–103
  - confirmatory test for, 99
  - development of, 95, 96f
  - drop arm test for, 99, 100f
  - factors leading to, 58–59, 62f
  - forearm drop test for, 99, 101f
  - magnetic resonance imaging of, 99
  - mechanisms leading to, 95–96
  - pain with, 97
  - partial or complete, 97–99
  - physical findings with, 97
  - shrugging mechanism with, 71
  - and suprascapular nerve entrapment, 190
  - treatment of, 100–103

Rotator cuff failure, and suprascapular nerve entrapment, 190
Rotator cuff tendon, 10, 25–26. *See also* Critical zone
- entrapment, 154

Rounded shoulder posture
- effect on glenohumeral range of motion, 63, 64f
- effect on shoulder anatomy, 124–126, 125f
- effects of, 57–58, 58f

Running
- leg gravity angle during, 156–157, 161f
- posterior shoulder flexibility and, 156–157

Scalene anticus syndrome, 178, 182
  Adson test in, 183–184, 184f
  physical examination in, 183–184
Scalene muscle(s), 170f, 178, 179f–180f
Scapula
  abductor rotator muscles, 45–46
  elevation of, with clavicular rotation, 40, 42f
  functional anatomy of, 3, 9, 10f, 27, 33f, 33–34, 124, 198–201, 199f
  in hemiplegia, 201, 202f–203f, 214, 217, 219
  mobility and strengthening exercises, 87, 92f
  muscles of, in hemiplegia, 204–205, 205f
  rotator muscles, 34, 34f–35f
  sites of muscle origin and insertion upon, 19f
  static support of, 40, 41f
  in upper extremity function. *See* Scapulohumeral rhythm, scapular component of
Scapulocostal joint, functional anatomy of, 2f, 2–3, 33f, 36
Scapulocostal syndrome, 253–257
Scapulohumeral rhythm, 2
  definition of, 27–28
  humeral phase of, 27–33
  scapular component of, 33–38
Scapulohumeral rhythm movement, 42–46, 45f
  accessory, other than glenohumeral movement, 45, 47f–48f
  ratio of humeral movement to scapular movement in, 43–44, 45f
Scapulothoracic joint, 3
  and posture, 46
  as site of pain or malfunction, 53, 53f
Semispinalis capitis muscle, 180f
Semispinalis cervicis muscle, 180f
Sensory fibers
  Ia, 19–21, 21f–22f
  II, 19–21, 21f–22f
Serratus anterior muscle
  functional anatomy of, 34, 34f, 36
  function of, 45–46
Sharpey's fibers, 202
Shoulder
  appearance, with dislocation, 141–142
  range of motion, exercises to increase, 85f–86f
Shoulder dislocation, 135–144
  anterior
    avenue of, 135, 136f
    mechanism of, 138, 138f
    primary, 135
    recurrence, 135–136
  diagnosis of, 141–142
  intra-articular fractures in, 139–140, 141f
  mechanism of, 137–141
    and age of patient, 137–138, 138f
    hyperextension theory, 138–139, 139f
  posterior. *See also* Shoulder dislocation, subspinous
    mechanism of, 140–141
  primary, mechanism of, 139
  recurrent, mechanism of, 139
  rotator cuff tear in, 96
  subclavicular, 136, 137f
  subcoracoid, 136, 137f
  subglenoid, 136, 137f
  subspinous, 136, 137f. *See also* Shoulder dislocation, posterior
  treatment, 142–144
  types of, 136–137
Shoulder dysfunction, roentgenographic changes in, 56, 57f
Shoulder girdle complex, definition of, 1
Shoulder girdle muscles, 19f
  strengthening exercises for, 84–87, 90f–91f
Shoulder girdle pain, 253
  articular, sites and causes of, 53
  musculoskeletal, sites and causes of, 51
  neurologic, sites and causes of, 51–52
  referred visceral-somatic, sites and causes of, 52–53
  vascular, sites and causes of, 52
Shoulder-hand-finger syndrome, 113, 235–251. *See also* Reflex sympathetic dystrophy
  arterial component, 235
  blood flow in, 240–241
  diagnosis, 237–241
  diminution of finger flexion in, 238–239, 239f
  finger changes in, 238–239, 239f, 241
  frozen, sequence leading to, 238f
  hand edema in, 238–241
    removal of, 245–247, 246f–247f
  hyperhidrosis in, 240–241
  hypertrichosis in, 241
  mechanism of, 237
  onset, 238, 241
  osteoporosis in, 241–242
  pain from, 236
  restoration of range of motion in, 245–249
  sequelae, 248
  stages of, 238f, 241–243
  treatment, 243–249
  vasomotor-sudomotor abnormality in, 240–241
  venous component, 235–236
Shoulder joint
  definition of, 1
  functional anatomy, 1–50
Shoulder limitation, signs of, 110, 111f
Shoulder pointer, 145
Shoulder tendinitis. *See* Tendinitis

Shrugging mechanism, 71, 73f–74f
  in frozen shoulder, 110
  in hemiplegia, 217
Side bending exercises, 164f–165f
Sleep disturbances, and fibrositis, 259–260
Sling(s), 75
  for treatment of shoulder subluxation with hemiplegic shoulder, 211–212, 213f–215f
Spica cast, for rotator cuff tear, 103, 103f
Spinal accessory nerve, 34
Spinal curves, development of, 128–130
Spinal motor centers, 195–196, 197f
Spine, lateral bending of, in hemiplegia, 201, 202f–203f
Splenius capitis muscle, 180f
Splenius cervicis muscle, 180f
Splint, air-inflated, 207, 208f
Sports injuries, 149–167
  and age, 149–150
  diagnosis, 154–155
  pain of, management, 159–160
  tests for, 155
  treatment, 157–167
Spurling compression test, 174, 177f
Standing tall, 133f
Stellate block, for reflex sympathetic dystrophy, 243–245, 244f
Sternoclavicular joint
  functional anatomy of, 2f, 2–3, 40–41, 41f, 43f
  palpable trigger point, 70f
  as site of pain or malfunction, 53, 53f
Sternoclavicular ligaments, functional anatomy of, 41
Sternocleidomastoid muscle, 180f
  functional anatomy of, 41–42, 44f
Sternocostal joint, 2f, 2–3
Sternocostovertebral articulations, 3
Sternum, 3, 9
Stiff shoulder, causes, 237
Stratum fibrosum, 202
Stratum synovium, 202
Stress, abnormal, 68–69
Stress management, for thoracic outlet syndrome, 186
Stroke. *See also* Cerebrovascular incident
  completed, 193–198
    definition of, 194
  functional recovery after, 206–207
  sensory loss in, 205–206
  shoulder in, treatment, 207–209
Subacromial bursae, 17
  functional anatomy of, 67–68, 68f
  as site of pain or malfunction, 53f
Subacromial bursitis. *See* Subdeltoid (subacromial) bursitis
Subclavian artery, 177, 179f
Subclavian vein, 177, 179f
Subcoracoid bursa, 17
Subdeltoid bursa
  functional anatomy of, 50, 55f, 67–68, 68f
  palpable trigger point, 70f
  as site of pain or malfunction, 53f, 54
Subdeltoid (subacromial) bursitis, 67–93, 114–116
  diagnosis, 68–73
  functional disability with, 71, 72f
  pain sites in, 70, 70f
  pain with, upon motion of arm, 71, 72f
  physiologic effect of exercise on, 87–93
  primary, 68
  radiologic diagnostic tests, 71–73
  shrugging mechanism with, 71, 73f
  strengthening exercises for, 84–87, 90f–91f
  therapeutic exercises for, 81–84
  therapy, 73–81
  trigger points, 70, 70f
Subluxation
  apprehension and relocation tests for, 155, 156f
  with hemiplegic shoulder, 209–212
  imaging modalities for, 155
  occult, 136–137
    diagnosis of, 141
Subscapular artery, 25, 27f
Subscapularis muscle
  action of, 31f
  calcium deposits in, 66, 71
  in frozen shoulder, 108
  functional anatomy of, 15, 18, 23–24, 24f–25f, 41–42, 44f
  function of, 32f, 33, 33f, 45
  in hemiplegia, 203, 204f, 214, 217
  insertion, palpable trigger point, 70f
Subscapular nerve, 24
Subscapular tendon, 24, 25f
Suprahumeral injection(s), 79–81, 80f
Suprahumeral joint
  functional anatomy of, 2f, 2–3, 11, 12f, 16–18, 39f, 55f
  periarticular case of, 109f
  as site of pain or malfunction, 53, 53f
Suprahyoid muscle, 180f
Suprascapular artery, 25, 27f
Suprascapular nerve, 18, 23
  block, 190f
  course of, 189, 190f
  entrapment, 189–191
  injection technique for, 81, 82f
Supraspinal motor centers, 195–196, 197f
Supraspinatus fossa, 10f
Supraspinatus muscle
  degeneration, impairment, fatigue, or pain, 54–56
  functional anatomy of, 10, 10f, 14, 18, 19f–20f, 22–23, 23f–24f, 200
  function of, 28–30, 30f–32f, 45

  in hemiplegia, 201, 203, 203f
  muscle spindle system, 18–22
  spindle system control of, 200, 200f
  strengthening exercises for, 84–87, 90f–91f
Supraspinatus tendinitis, 114–116. *See* Subdeltoid (subacromial) bursitis
  painful arc in, 71, 74f
  symptoms of, 60–63
  therapy, 73–81
Supraspinatus tendon
  blood flow to, 92
  calcium deposits in, 66, 71
  critical zone, 256–257. *See also* Critical zone
  degeneration, from ischemia, 92
  effects of aging on, 57
  functional anatomy of, 10, 17, 22, 23f, 50, 55f
  insertion, palpable trigger point, 70f
  pathology, 54–55
  as site of pain or malfunction, 54
  tears of, 63
  traumatic consequences on, 58–59
Sympathetic, definition of, 228
Sympathetically maintained pain, 228
  neurophysiological mechanism of, 231–233, 234f
Sympathetic independent pain, 228
Synovial fluid, 4, 5f
Synovium, 4

Temporomandibular pain and dysfunction syndrome, 258
Tendinitis, 54–60. *See also* Biceps tendinitis; Calcific tendinitis; Supraspinatus tendinitis
  pathogenesis, 56
  from repetitive contractions, 56
Tendon
  inflammation, from repetitive contractions, 92
  trauma to, 91–92
Tennis, injuries in, 154–155
Tennis serve, mechanics and tissues involved in, 150, 151f
TENS. *See* Transcutaneous electrical nerve stimulation
Tension myositis, 22
Teres major muscle, functional anatomy of, 18
Teres minor muscle
  action of, 29
  calcium deposits in, 66, 71
  functional anatomy of, 10, 18, 23, 24f
  function of, 32f, 45
  in hemiplegia, 203
Thoracic curve, development of, 128–129
Thoracic nerve(s), 36–37
Thoracic outlet
  definition of, 177, 182
  functional anatomy, 177, 179f
  neurovascular bundle, 177, 179f
Thoracic outlet syndrome, 176–186
  definition of, 178
  hyperabduction compression syndrome, 183
  operative intervention for, 186
  physical examination in, 183–185
  signs and symptoms of, 180–182
  treatment, 185–186
Thoracoscapular articulation, 34
Tissue ossification, in stroke patient, 223–224
Tissue trauma. *See* Trauma
Tonic neck reflexes, in hemiplegia, 214–215, 216f
Transcutaneous electrical nerve stimulation
  for causalgic pain, 245
  for treatment of frozen shoulder, 114, 118
Transient ischemic attacks, 193–194
Translation exercise, 131
Transverse humeral ligament, functional anatomy of, 24, 48
Trapezius muscle
  functional anatomy of, 34f, 34–36, 41–42, 44f
  function of, 45–46
  moth-eaten fibers, 93
  pain in, 253–257
Trapezoid ligament, functional anatomy of, 39–40, 40f–41f
Trauma
  brachial plexus injuries in, 186–188
  chemical mediation of, 58, 61f
  to tendon, 91–92
  vascular sequelae of, 58, 61f
Trigger point(s), 257–258
Trunk flexibility exercise, swinging warm-up, 162f
Trunk movement, in hemiplegia, 217
Trunk twist exercise, 163f
Type A personality, 250

Ultrasound, 114
Upper extremity. *See also* Arm
  definition of, 2
  flexor synergy of, 197, 199f
  functional anatomy of, 1–50
  venous lymphatic pumps of, 235–236, 236f

Vestibular system, 128
Voluntary muscular weakness, Bobaths' concept of, 217–219

Wall-climbing exercise, 87, 91f
Wheelchair support, for treatment of

    shoulder subluxation with hemiplegic shoulder, 212, 215f
Wide dynamic range neurones, 231–233, 234f
Wrist joints, mobilization, 247, 248f

about this series . . .

Dr. Cailliet's books have been applauded by doctors and health professionals all over the world; they provide commonsense guides for diagnosis and treatment of painful or disabling conditions in various parts of the body. One of the outstanding features of these books is the concise and detailed descriptions of the structure and functional anatomy of each region, with numerous drawings graphically expanding the author's lucid text.

These books have been acclaimed by readers worldwide. Some comments:

"... would recommend this series highly"
"... practical, readable, and understandable"
"... always a valuable and clear-cut approach" (to treatment)

the other titles:

LOW BACK PAIN SYNDROME (Edition 4)
336 pages/216 illustrations
KNEE PAIN AND DISABILITY (Edition 2)
177 pages/126 illustrations
FOOT AND ANKLE PAIN (Edition 2)
200 pages/134 illustrations
HAND PAIN AND IMPAIRMENT (Edition 3)
232 pages/167 illustrations
NECK AND ARM PAIN (Edition 3)
226 pages/185 illustrations
SCOLIOSIS: DIAGNOSIS AND MANAGEMENT
121 pages/62 illustrations
THE SHOULDER IN HEMIPLEGIA
130 pages/77 illustrations
SOFT TISSUE PAIN AND DISABILITY (Edition 2)
382 pages 277 illustrations
UNDERSTAND YOUR BACKACHE
194 pages/105 illustrations

0-8036-1614-7